Transient Hyperopia after Intensive Treatment of Hyperglycemia in Newly Diagnosed Diabetes

Shu-Fang Lin^{a,b} Po-Kang Lin^{c,d} Fang-Ling Chang^a Rong-Kung Tsai^{a,b}

^aDepartment of Ophthalmology, Buddhist Tzu-Chi General Hospital, and ^bDepartment of Ophthalmology and Visual Science, Tzu-Chi University, Hualien; ^cDepartment of Ophthalmology, Taipei Veterans General Hospital, and ^dDepartment of Ophthalmology, School of Medicine, National Yang-Ming University, Taipei, Taiwan, ROC

Key Words

Hyperglycemia · Transient hyperopia · Diabetes

Abstract

Purpose: Our aim was to observe the transient hyperopia during the intense glucose reduction in patients with newly diagnosed diabetes and severe hyperglycemia. **Study Design:** Consecutive cases were observed. **Results:** Totally 4 men and 1 woman with a mean age of 48 years were enrolled. In the 4 patients who received insulin, the hyperopia developed at 4.2 days after the initiation of treatment on average and reached a peak at 11.7 days; they recovered at 64.0 days. The other subject who received oral hypoglycemia agents revealed a peak change at 17 days and recovered at 70 days. A broader hyperopic change of 6.25 dpt was found in the patient with high myopia (–16 dpt). No significant difference was observed in the corneal curvature, axial length, lens thickness or depth of the anterior chamber during the course. The stable value of the accommodation amplitude and lens thickness may indicate that the cause of refraction change was due to the alteration in the reflection index of the lens. **Conclusion:** Intensive glucose reduction may cause transient hyperopia changes in newly diabetic patients and results in blurred vision.

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Introduction

Diabetic patients suffer from various ocular disorders such as cataract, ischemic optic nerve neuropathy and retinal vasculopathy [1–3]. It is well known that diabetic patients may have both short-term and permanent alterations in refraction status.

The refractive power of the eyes depends on the anterior and posterior cornea curvature, corneal thickness, anterior chamber depth (ACD), lens thickness (LT) and curvature, axial length (AL), as well as the refractive index of the cornea, aqueous, lens and vitreous [3].

The refraction change in diabetics was thought to be ‘diabetic myopia’ in 1925 by Duke-Elder [4], supported by the study of Gwinup and Villarreal [5] thereafter. However, Fledelius [6], Fledelius et al. [7] and Eva et al. [8] observed different refraction changes in diabetic patients with short- and long-term dysregulation; the refraction would become less myopic or more hyperopic while the plasma glucose concentration decreased rapidly. After the hyperopic change had appeared, the refractive normalization happened weeks behind the regulation of blood sugar. The changes in the surface curvature and the refractive index of the lens due to osmotic forces were thought to be the main cause. We collected 5 patients with new-onset diabetes mellitus with transient hyperopia after intensive hypoglycemic treatment and studied the relative optical indexes in their natural course.

Table 1. Clinical characteristics of the diabetic patients

Patient number	Gender	Age years	Duration of DM	DM type	Pretreatment FPS, mg/dl	HbA _{1c} %	DM retinopathy (OD/OS)
1	M	52	ND	NIDDM	406	15.5	-/-
2	F	58	ND	NIDDM	411	14.6	-/-
3	M	50	ND	NIDDM	800	NA	-/-
4	M	41	ND	NIDDM	450	15.2	-/-
5	M	39	ND	NIDDM	881	11.8	-/-

DM = Diabetes mellitus; FPS = fasting plasma glucose; ND = newly diagnosed; NA = not available.

Patients and Methods

The clinical characteristics of the patients are summarized in table 1. All of them were diagnosed as having new-onset non-insulin-dependent diabetes mellitus (NIDDM) and had a mean age of 48 years, ranging from 39 to 58. There were 4 male and 1 female patients. Four of the 5 subjects were admitted to receive strict blood sugar control with insulin administration. One patient received oral hypoglycemic agents (OHA) and was followed in the outpatient clinic. At all ophthalmic visits, refraction and corneal curvature (K) were measured with a Nidek ARK-700 automatic refractometer (Nidek Co. Ltd., Gamagori, Japan) and the corrected visual acuity was evaluated. The intraocular pressure was measured with a noncontact tonometer. A-mode ultrasonography was performed to measure the ACD, LT and AL, and an average of 10 measurements was recorded. Three cases (patients 1, 2 and 5) were followed on schedule and had more complete data; the other 2 had data at the peak and recovery stages. The change in accommodation amplitude of case 5 was checked by Jacques blur point card and cross cylinder at every weekly follow-up. Fundus pictures were taken and fluorescein angiography was performed in the inward patients to evaluate the possible retinopathy.

Case 1

A 52-year-old man with newly diagnosed diabetes mellitus was placed on OHA for hyperglycemia with a preprandial value of 406 mg/dl. Ten days later, the patient called for help because of bilateral blurred vision. On ocular examination, the best-corrected visual acuity was 6/7.5 with +2.25 dpt in the right eye and 6/10 with +2.0 dpt in the left. The intraocular pressure, anterior segment and fundus were all normal. The hyperopic shift reached its peak on the 17th day after blood sugar control (OD +3.25, OS +3.0/ cyl -0.5 × 15°) and regained the original level gradually on the 72nd day (OD +0.25, OS -0.5). Serial examinations showed no significant change in AL, ACD, average K and LT. The best-corrected visual acuity remained stable throughout the course.

Case 2

A 58-year-old woman with newly diagnosed diabetes mellitus was admitted to receive aggressive hyperglycemic control. On admission, her corrected visual acuity was 6/7.5 in both eyes. Seven days later, she noticed blurring of near vision. Ophthalmic ex-

Fig. 1. The refraction change patterns and the sugar change of case 1 (a) and case 2 (b). The peak amplitude of hyperopic treatment happened around the second and third weeks after hypoglycemic treatment and the refractive shift improved at 3 months after treatment.

aminations revealed normal results except for the hyperopic shift in refraction. During the following visits, the maximum hyperopic change reached 3.0 (OD) and 3.5 dpt (OS) on the 14th day and began to improve gradually. By the 90th day, her refraction status regained the original level. The AL, ACD, average K and LT showed no significant change throughout the following period either (fig. 1).

Table 2. Clinical course of transient hyperopic change

Patient number	Onset days	Peak days	Recovery days	Baseline refraction (OD/OS) dpt	Max. change (OD/OS) dpt
1	10	17	72	+0.25/-0.5	+3.0/+3.5
2	7	14	90	+2.25/+2.0	+3.5/+3.25
3	7	10	61	plane/+0.25	+3.5/+3.5
4	2	14	65	-14.25/-16.25	+6.25/+6.25
5	1	9	40	+0.5/+0.25	+2.75/+4.75

Case 1 received OHA in the outpatient clinic.

Table 3. The change in refractive components of all patients

	At peak of hyperopic change	Recovery of hyperopic change	p
Anterior chamber depth, mm	3.20 ± 0.50	3.21 ± 0.54	NS
Lens thickness, mm	4.40 ± 0.30	4.26 ± 0.34	NS
Axial length, mm	24.29 ± 2.70	24.23 ± 2.70	NS

NS = No significant difference.

Case 3

A 50-year-old man with diabetes mellitus was admitted to control the high blood sugar. The patient had suffered from blurred vision for about 1 week and presented with an initial fasting plasma glucose of 800 mg/dl. After intensive blood sugar control, the level fell to 250 mg/dl and a hyperopic shift of 3.5 dpt was noted bilaterally during the admission course (from OD plane/cyl -0.25 × 5°, OS +0.25/cyl -0.5 × 175° to OD +3.5, OS +3.75/cyl -0.5 × 180°). The AL, ACD, average K and LT showed no significant change.

Case 4

A 41-year-old man with high myopic status (OD -14.25/cyl -1.75° × 180, OS -16.25/cyl -0.75 × 180°) had been diagnosed as having NIDDM for about 2 weeks. The blood sugar was 450 mg/dl while admitted. He felt blurred vision when he was wearing his glasses 2 days after the onset of insulin treatment. The ophthalmologic examination showed: the best-corrected vision was 6/10 with -8.5/cyl -2.0 × 180° and 6/6.7 with -10.5/cyl -0.75 × 175°. A maximum hyperopic shift of 6.25 dpt was noted in both eyes. The AL were 28.83 mm OD and 29.55 mm OS, and the ACD were 3.65 mm OD and 3.94 mm OS. Two months later, the refraction regained the original level. The AL, ACD and LT remained unchanged throughout the course.

Case 5

A 39-year-old man suffered from blurred distant vision and was accidentally found with a fasting plasma glucose of 881 mg/dl. The initial ophthalmologic evaluation showed: the best-corrected

vision was 6/6 with -3.0 dpt OD and 6/6 with -3.0 dpt OS. After intensive blood sugar control, the blood sugar level fell to 180 mg/dl in 10 days and a maximum hyperopic shift of 4.75 dpt was recorded on the 9th day. The AL was 23.9 mm OD and 24.1 mm OS. Forty days later, the refraction regained the original level (±0.5 dpt OD, ±0.25 dpt OS). The AL, ACD and LT remained unchanged throughout the course. The accommodation amplitude was 4.66 dpt OD and 3.41 dpt OS at the peak, and 4.2 dpt OD and 3.25 dpt OS in the final follow-up.

Results

The clinical courses of the transient hyperopic changes are shown in table 2. At the beginning of treatment, the mean fasting plasma glucose value was 589.6 ± 231.4 mg/dl (range: 406–881) and the mean HbA_{1c} value was 14.2% (range: 11.8–15.5).

The transient hyperopic changes happened in all the patients after sugar control. In the 4 cases receiving insulin, the hyperopic change appeared at 4.2 ± 3.8 days after treatment initiation and reached a peak at 11.7 ± 3.3 days. The achievement of the original refraction occurred around 64.0 ± 18.1 days. The patient who received OHA treatment demonstrated a peak change on day 17 and recovered on day 72 with a maximum refraction change of 3.0 dpt. The largest hyperopic shift was 6.25 dpt in the high myopia patient (case 4). During the transient hyperopia period, no significant changes were found in the K, AL, LT or depth of the anterior chamber (table 3). No significant variation in the accommodation amplitude occurred during the course, which revealed that the accommodation power had no correlation with the hyperopic shift. None of the patients developed diabetic retinopathy throughout the course.

Discussion

Several reports have described the refractive changes associated with elevated or depressed blood glucose levels [4–9]. The correlation between the maximum hyperopic change of an eye and sex, age, duration of disease and type of hypoglycemic therapy was proved to be nonsignificant in the study by Okamoto et al. [10].

In the glucose loading study on healthy human volunteers, the myopic change was explained by the thickening of the lens, which led to a decrease in the tension of the zonule fibers of Zinn; moreover, in the normalization course of plasma glucose, a hyperopic reversal phenomenon was revealed [11]. Saito et al. [12] also reported tran-

sient hyperopia in diabetic patients after glycemic control. A mean change of 3 dpt was found. During the hyperopia period, there were no significant alterations in the AL or K. However, a thickened lens, decreased ACD and transient cataract were observed in their report. They proposed that the lens swelling due to water influx induced a lower lens refractive index and the following hyperopia.

However, Planten [13] and Planten et al. [14] found no significant difference in the change in LT. In the study of Le Grand's full theoretical eye [14], they demonstrated that a decrease in the refractive index of the lens from 1.42 to 1.40 would produce a hyperopic change of 3.2 dpt. They thought that the lens refractive index itself might be responsible for the refraction change.

Tai et al. [15] also confirmed a stability of optic components except the reduction in the refractive index of the lens during the refractive fluctuation in the diabetic patients. In their study, 34% of the long-term NIDDM patients who received insulin to control hyperglycemia revealed a hyperopic change during the treatment course.

In the report by Okamoto et al. [10], there were 4 patients with newly diagnosed NIDDM who received OHA to control hyperglycemia. In their study, the initial blood glucose was 506.7 ± 29.4 mg/dl. The hyperopic change appeared at 3.25 ± 2.8 days after treatment and reached a peak at 10.5 ± 4.1 days. The achievement of the original refraction occurred around 49 ± 27.4 days. The magnitude of hyperopic refraction change was 1.35 ± 0.5 dpt. Our patient who received OHA treatment demonstrated a peak change on day 17 and recovered on day 72 with a maximum refraction change of 3.0 dpt.

In the study by Okamoto et al. [10], there seems to exist a slight delay in the refraction change pattern in the

insulin treatment group. However, the number of patients is not large enough to draw a conclusion.

Okamoto et al. [10] demonstrated a positive correlation between maximum hyperopic change and the baseline refraction status. The greater the degree of myopia, the larger the hyperopic change occurring during strict sugar control. They proposed the hypothesis that myopic eyes have a larger volume with a long AL and a dysfunctional blood-ocular barrier; and the changes in the compositions of the intraocular fluid and osmotic pressure would be greater. In our study, the high myopic patient also revealed a maximum hyperopic change of 6.25 dpt, compatible with the hypothesis.

In our evaluation, 2 cases with high blood sugar (800 and 881 mg/dl) had a relatively larger hyperopia change (3.5 and 4.75 dpt) during the course. A positive relationship between the initial plasma glucose concentration and the magnitude of the maximum hyperopic change has been reported ($r = 0.57$) [10].

Conclusion

We demonstrated that hyperopic refraction changes occurred in patients with newly diagnosed NIDDM in a rapid hypoglycemic process. Our study showed that most of the optical components of the eyes remained stable. The constant values of accommodation amplitude and LT may indicate that the refraction change was due to the variation in the reflection index of the lens. Further study of optic factors related to transient hyperopia in diabetic patients on hypoglycemic treatment is necessary to clarify the mechanisms.

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