#### ·Clinical Research·

# Effects of glycemic control on refraction in diabetic patients

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## Abstract

• AIM: To evaluate the effects of glycemic control on refraction in diabetic patients.

• METHODS: Twenty newly diagnosed diabetic patients were included in this study. The random blood glucose, HbA1c levels, fasting C-peptide and postprandial 2h C-peptide were measured before treatment. The patients with random blood glucose higher than 12.0mmol/L and HbA1c level higher than 10.0% were selected. Refraction, intraocular pressure, radius of the anterior corneal curvature, depth of the anterior chamber, lens thickness, vitreous length, and axial length were measured on admission and at the end of week 1, 2, 3 and 4 during glycemic control.

• RESULTS: A transient hyperopic change occurred in all the patients receiving glycemic control. The maximum hyperopic change was 1.60D (range  $0.50 \pm 3.20D$ ). Recovery of the previous refraction occurred between two and four weeks after insulin treatment. There was a positive correlation between the maximum hyperopic changes and the HbA1c levels on admission (r=0.84, P<0.05). There was a positive correlation between the maximum hyperopic changes and the daily rate of blood glucose reduction over the first 7 days of the treatment (r=0.53, P<0.05). During transient hyperopia, no significant changes were observed in the intraocular pressure, radius of the anterior corneal curvature, depth of the anterior chamber, lens thickness, vitreous length and axial length.

• CONCLUSION: Transient hyperopic changes occur after glycemic control in diabetic patients with severe hyperglycemia. The degrees of transient hyperopia are highly dependent on HbA1c levels before treatment and the rate of reduction of the blood glucose level.

• KEYWORDS: refraction; diabetes mellitus; lens; blood glucose DOI:10.3980/j.issn.2222-3959.2010.02.15

# INTRODUCTION

 $\mathbf{D}$  uring hypoglycemic treatment, some diabetic patients suffer from blurred vision. It is well known that changes of plasma glucose lead to transient refractive error, but the biological basis of refractive changes in the eyes of diabetic patients has not yet been established and the underlying mechanism is still unknown. The authors conducted a prospective study of 40 eyes of 20 diabetic patients who underwent glycemic control for severe hyperglycemia, in an attempt to make an objective evaluation of refractive changes during treatment.

## MATERIALS AND METHODS

**Subjects** The study was conducted on 40 eyes of 20 newly diagnosed diabetic patients who had a plasma glucose level of 12.0mmol/L or higher, and a glycosylated haemoglobin (HbA1c) of 10.0% or higher on admission. The subjects consisted of 12 men and eight women, with a mean age of 46 years (range 28-58 years).

**Methods** Blood pressure, body mass index, triglyceride, total cholesterol, low-density lipoprotein, high-density lipoprotein, fasting C-peptide and postprandial 2h C-peptide were measured on admission (Table 1). All patients received insulin treatment to reduce blood glucose.

An ophthalmological examination was conducted on admission and at the end of week 1, 2, 3 and 4 during glycemic control. Refraction, intraocular pressure and radius of the anterior corneal curvature were measured in all subjects. A-mode ultrasonography was performed by the same examiner three times in each eye at each examination to measure the lens thickness, depth of the anterior chamber, vitreous length and axial length. The mean value of the three measurements was used for analysis of each biometric parameter. A change in refraction was defined when the refractive change was 0.5 dioptre or more compared with the value on admission. Blood glucose was measured at least four times at the seven day of hypoglycemic treatment. The mean blood glucose value at the seven day (BG7) was calculated. The daily rate of reduction of blood glucose concentration over the first 7 days of treatment was calculated by the following equation:  $\triangle BG = (BG0-BG7)/7$ . Statistical Analysis The results were presented as mean, and performed with paired Student's t test and simple regression analysis. P<0.05 was considered to be statistically significant.

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Table 1 Clin	(mean±SD)				
Age(years)	Blood glucose(mmol/L)	HbA1c(%)	F-CP(ng/mL)	P-CP(ng/mL)	TG(mmol/L)
45.50±8.10	19.48±5.04	12.20±1.50	1.25±0.41	2.53±0.89	2.34±1.19
TC(mmol/L)	LDL-C(mmol/L)	HDL-C(mmol/L)	BMI(kg/m <sup>2</sup> )	SBP(mmHg)	DBP(mmHg)
4.75±0.72	2.88±0.62	0.90±0.11	24.60±2.90	128.80±18.60	77.30±13.60

F-CP: Fasting C-peptide; P-CP: Postprandial 2h C-peptide; TG: Triglyceride; TC: Total cholesterol; LDL-C: Low-density lipoprotein; HDL-C: High-density lipoprotein; BMI: Body mass index; SBP: Systolic blood pressure; DBP: Diastolic blood pressure (1mmHg=0.133kPa)

Table 2 Ophthalmic parameter		$(\text{mean}\pm\text{SD})$		
	Before hypoglycemic treatment		The peak time of hyperopic change	
-	R	L	R	L
Intraocular pressure (mmHg)	15.01±2.77	15.30±2.90	$15.08 \pm 2.60$	15.28±2.83
Corneal curvature radius (mm)	7.55±0.27	7.58±0.19	7.51±0.18	7.54±0.14
Lens thickness (mm)	4.34±0.15	4.36±0.20	4.35±0.15	4.39±0.16
Anterior chambe depth (mm)	3.34±0.08	$3.35 {\pm} 0.08$	3.36±0.08	3.42±0.10
Vitreous length (mm)	16.48±0.13	16.49±0.11	16.50±0.14	$16.54 \pm 0.14$
Axial length (mm)	23.84±0.62	23.80±0.66	23.86±0.57	23.84±0.60

R:Right eye; L:Left eye; P<0.05

0.14

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## RESULTS

The mean random blood glucose at admission (BG0) was 19.5mmol/L (range 12.0-28.6mmol/L) and the mean HbA1c value was 12.2% (range 10.1% -15.3%). A transient hyperopic change occurred in all diabetic patients receiving hypoglycemic treatment. The maximum hyperopic change occurred at the end of the first week in 10 patients, at the end of the second week in 7 patients and at the end of the third week in 3 patients following treatment. A mean maximum hyperopic change was 1.60 diopters(range 0.50-3.20 diopters). Refraction gradually returned to the baseline value at the end of the second week in 3 patients, at the end of the first week in 7 patients. Refraction did not yet returned at the end of the fourth week in 7 patients. Refraction did not yet returned at the end of the fourth week in one patient, and the patient was lost to follow then.

There was a significant positive correlation between the maximum hyperopic change and HbA1c level on admission (r = 0.84, P < 0.05, Figure 1). There was also a significant positive correlation between the maximum hyperopic change and  $\triangle$  BG (r=0.53, P < 0.05, Figure 2). There was no significant correlation between the maximum hyperopic change and random blood glucose on admission (BG0), fasting C-peptide, postprandial 2h C-peptide, age, blood press, body mass index, triglyceride, total cholesterol, low-density lipoprotein and high-density lipoprotein (P > 0.05).

The intraocular pressure, radius of the anterior corneal curvature, depth of the anterior chamber, lens thickness, vitreous length and axial length at the peak time of hyperopic change did not differ from the respective value measured before treatment was started (P>0.05, Table 2).

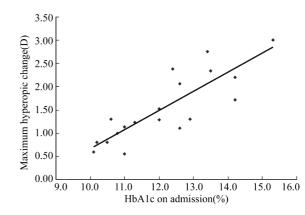


Figure 1 Relation between maximum hyperopic change and HbA1c levels on admission There was a significant positive correlation between them (r=0.84, P<0.05)

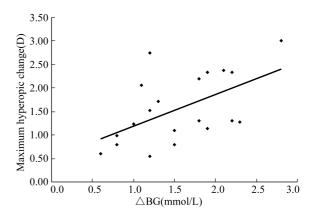


Figure 2 Relation between maximum hyperopic change and the daily rate of glucose reduction over the first 7 days of treatment There was a positive correlation between them(r=0.53, P<0.05)

### DISCUSSION

It has been reported that decreasing plasma glucose level causes hyperopic change <sup>[1-3]</sup>. However, some investigators have observed both myopic and hyperopic changes in diabetic eyes <sup>[4,5]</sup>. The mechanism of alteration in blood glucose concentration leading to refractive change in diabetics is not yet clear. The present study investigated the clinical course of the refractive change of 40 diabetic eyes during glycemic control. This study revealed that during treatment of hyperglycemia in diabetic patients, a transient hyperopic change of 0.5 diopter or more developed in all eyes. Hyperopic change gradually returned to the baseline value within four weeks in most patients. After receiving hypoglycemic treatment, some diabetic patients complain of blurred vision because of refractive change and new glasses are prescribed. However, new glasses will not fit after 2 or 4 weeks. So doctors should explain the refractive change is transient and the prescription of new glasses should be delayed. If glasses must be prescribed during the occurrence of transient hyperopia, further modifications in the prescription may be needed when the refraction returns stable.

In the present study, patients who had a higher HbA1c on admission had a larger maximum hyperopic change. There was a definite positive correlation between the maximum hyperopic change and the daily rate of plasma glucose reduction over the first 7 days during glycemic control. However, there was no significant correlation among the maximum hyperopic change and each of age, body mass index, blood press, lipid, and C-peptide level. These results indicate that the degree of hyperopia is highly dependent on the rate of plasma glucose reduction and the degree of hyperglycemia. No significant correlation was observed between the maximum hyperopic change and random blood glucose on admission (P=0.06). It is probably because that HbA1c is more accurate than random blood glucose in reflecting hyperglycemia degree.

Lens abnormalities have been suggested as a cause of refraction error in diabetic patients. Excess glucose in the lens is converted to sorbitol during hyperglycemia. Sorbitol is poorly permeable and accumulates in the lens. When blood glucose reduces, the difference in osmotic pressure results in the influx of water from the aqueous humour into the lens, causing lenticular swelling <sup>[36,7]</sup>. However, an increase in lens thickness would promote myopic changes through increase of refractive power, which seems to be contradictory with the observation of hyperopic changes when plasma glucose reduces. The hypothesis is that lens cortex hydration following the influx of water from the aqueous humour results in the decrease of refractive power, which might be involved in the development of transient hyperopic change. The morphological change of the lens is minor. The present study found no significant changes in the lens thickness, depth of the anterior chamber and axial length, which supports the above hypothesis.

Overall, a transient hyperopic change occurred in diabetic patients during glycemic control. The degree of hyperopia is highly dependent on the HbA1c level before treatment and the rate of plasma glucose reduction over the first 7 days of treatment. It might be the mechanism that lens cortex hydration results in the decrease of refractive power, not that the morphology of lens changes.

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