Acute Incapacitating Hyperopia during Hypoglycemic Treatment

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Introduction

Patients with diabetes mellitus can develop various ocular complications such as cataract, retinopathy, optic neuropathy, uveitis, and keratopathy etc. In addition, transient refractive error occurs during the course of diabetes mellitus and is associated with treatment induced changes in the plasma glucose concentration [1,2]. The association of myopia with hyperglycaemia and hyperopia with hypoglycaemia have been reported in diabetic patients [3]. There are studies which show that an abrupt reduction in plasma glucose in diabetic patients with marked hyperglycaemia, induce transient hyperopia [1,2,4]. It has been seen that the degree of hyperopia is highly dependent on the magnitude of the change in plasma glucose concentration [2]. Patients may get alarmed and may request for change of glasses but fresh prescription should be given only when the refraction stabilises, otherwise, the glasses thus prescribed will soon become inadequate. A case is presented where a lady developed acute incapacitating hyperopia during hypoglycemic treatment. It is quite unusual to come across such high degree of induced transient hyperopia in clinical practice. The pattern of development and gradual decay of the transient hyperopia is described with review of relevant literature.

Case Report

A 40 year old lady developed polyurea, polydipsia and polyphagia. Her detailed general and systemic examination was found normal. Her blood sugar levels were found to be, fasting 310 mg/dl and postprandial 504 mg/dl. All other relevant investigations were found to be normal. She was diagnosed as a case of diabetes mellitus and was put on a diabetic diet, glibenclamide, and metformin.

Her earlier prescription of glasses was, +0.50D sph for right eye, +0.75D sph for left eye for distant vision and an addition of +1.00 D for near vision resulting in 6/6 and N5 vision in both eyes respectively. Her vision started deteriorating on the fifth day of treatment and on the seventh day it became incapacitating. She reported to the eye department on the seventh day of treatment and requested for change of glasses. Detailed ophthalmological examination

was conducted. Corneas were clear, pupils were normal, and there was no cataract and funduscopy didn't reveal retinopathy or macular oedema. Intra ocular pressure was normal. Refractive status of the eye was checked and was followed up for next few weeks. It was found that the patient developed hyperopia which started on fifth day of treatment, reached its peak at +3.00D above the baseline refraction, and gradually came back to the baseline level by the end of seventh week. Through these seven weeks the patient was reassured and no change of glasses was advocated. Finally the patient became comfortable with her previous glasses. The details of change of refraction are given in Table 1.

Discussion

Refractive changes are known to be associated with diabetes mellitus and these can be acute or chronic. Regarding chronic refractive changes in diabetic patients, Duke-Elder reported that hyperglycaemia led to the development of myopia, while hypoglycaemia led to the development of hyperopia [3]. Many authors, who investigated the effect of acute changes in plasma glucose level, have reported that decreasing plasma glucose levels cause hyperopic changes [1,5,6]. Although some conflicting reports are there, recently, several papers have reported an abrupt reduction in plasma glucose in diabetic patients with marked hyperglycaemia induced transient hyperopia [1,2,4]. It has been seen that the degree of hyperopia is highly dependent on the magnitude of the change in plasma glucose concentration [2].

Lenticular swelling has been suggested as a cause of hyperopia in diabetic patients. In patients with diabetes mellitus, excess glucose in the crystalline lens is converted to sorbitol through the action of aldose reductase. Sorbitol, a sugar alcohol, has poor permeability through membranes and tends to accumulate in the lens [7]. When the body rapidly changes from a hyperglycaemic to a hypoglycaemic state, sorbitol, which is less permeable and harder to metabolise, will remain in the lens longer. The difference in osmotic pressure results in the influx of water from the aqueous humour into the lens, causing lenticular

Table 1

Details of the change of refraction during hypoglycemic treatment

Day of treatment	Acceptance of glasses for distance		Acceptance of glasses for near		Blood sugar level in mg/dl	
	Right eye	Left eye	Right eye	Left eye	Fasting	Post prandial
Before treatment (baseline)	+0.50D	+0.75D	+1.5D	+1.75D	310	504
7	+2.25D	+2.50D	+3.25D	+3.50D	124	196
13 (peak)	+3.50D	+3.75D	+4.50D	+4.75D	102	169
22	+3.50D	+3.75D	+4.50D	+4.75D	-	-
28	+2.00D	+2.25D	+3.00D	+3.25D	106	162
36	+1.25D	+1.50D	+2.25D	+2.50D	114	158
49 (baseline)	+0.50D	+0.75D	+1.50D	+1.75D	111	154

swelling with hyperopic refractive changes. This is a possible hypothesis for explaining the occurrence of transient hyperopic changes which is supported by Saito et al [1], whereas, debated by Okamoto [2].

An experimental study demonstrated that a decrease in refractive index of the lens from 1.42 to 1.40 produced a hyperopic change of 3.2 dioptres; therefore, a slight change in the refractive index produces a significant hyperopic change [8]. Gwinup and Villarreal [6] further proved that hyperopic changes are primarily due to lens. So, it appears that a change in the refractive index of the lens plays a significant role in the development of transient hyperopia.

In clinical practice it has been seen that some diabetic patients may complain of blurring of vision due to development of transient hyperopia during intensive hypoglycaemic treatment. Saito et al [1], in a study of 10 eyes of five diabetic patients, found that bilateral transient hyperopia with lenticular swelling occurred after initiation of strict hypoglycaemic control. Okamoto F et al [2] studied 28 eyes of 14 diabetic patients during intense hypoglycemic treatment. He found that a transient hyperopic change occurred in all patients receiving improved control after hyperglycaemia. Hyperopic change appeared 1-7 days after treatment was started; reached its peak after 4-28 days; and returned to the baseline value over 14-84 days, with a mean maximum hyperopic change of 1.47 dioptres in the 28 eyes. During transient hyperopia, no significant changes were observed in the radius of the anterior corneal curvature, axial length, lens thickness, or depth of anterior chamber.

Our patient had developed quite high hyperopia of 3D above her baseline refraction, which is quite unusual. The other causes of visual deterioration in diabetic

patients are progression of retinopathy, macular oedema and diabetic maculopathy. Our patient had none of these and she did not suffer a decline in the corrected visual acuity.

After beginning intensive glycaemic control for hyperglycaemia, diabetic patients may get worried by the blurred vision and request a prescription of new glasses. However, if possible, the prescription of spectacles should be delayed until a stable refraction is obtained. If glasses must be prescribed while the refractive status is still unstable, the patient should be informed that the refraction may change over time and that further modifications in the prescription may be needed.

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