Corneal Abnormalities in Diabetes†

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Abstract

Objective : To compare corneal thickness and endothelial morphology in patients with diabetes mellitus and age-matched normal subjects, and to determine whether the duration of diabetes mellitus, severity of diabetic retinopathy, and glycemic control are correlated with these measurements.

Design : Single center, case-control study.

Participants : Sixty eyes of thirty diabetic patients and sixty eyes of thirty healthy non-diabetic subjects were studied.

Intervention : Corneal thickness was measured by ultrasonic pachymeter. Corneal endothelial morphology was examined with a contact specular microscope.

Main Outcome Measures : Corneal endothelial cell density, mean cell area, coefficient of variation, percentages of hexagonal cells, and corneal thickness were measured.

Results : There was statistically significant increased corneal endothelial cell density and decreased mean endothelial cell area in the diabetic patients. The diabetic corneas had an increased coefficient of variation of endothelial cell area, a decreased percentage of hexagonal endothelial cell and an increased corneal thickness compared with the control subjects, but these differences were not statistically significant. The duration of diabetes mellitus was significantly correlated with pleomorphism, polymegathism and corneal thickness. Severity of diabetic retinopathy was correlated with endothelial cell density, but these correlations were low. The corneal changes were not correlated with glycemic control.

Conclusions : The diabetic corneas tended to be thicker and had more pleomorphism and polymegathism, though this was not statistically significant. Duration of diabetes mellitus correlated

significantly with these corneal changes. This suggests that corneal changes should be evaluated and confirmed before intraocular surgery in chronic diabetic patients.

Key word : Corneal Endothelial Cell Density, Coefficient of Variation, Pleomorphism, Polymegathism, Corneal Thickness, Diabetes Mellitus.

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J Med Assoc Thai 2001; 84: 1075-1083

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† Presented at the Royal College of Ophthalmologists of Thailand Annual Meeting, Cha-am, Petchburi, July 16, 2000.

Clinical observations in patients with diabetes mellitus have revealed a higher incidence of delayed epithelialization, persistent epithelial defects, recurrent epithelial erosions, stromal edema after pars plana vitrectomy, reduced corneal sensation, neurotrophic corneal ulcers, wrinkles in Descemet's membrane and various corneal endothelial abnormalities[1-13].

The endothelial cell density in a normal adult cornea is in the range of 1500-3500 cells/mm^2 with coefficient of variation of cell area (CV) 25-31 per cent, hexagonal cells 61-74 per cent, and corneal thickness 527-570 μ[14-22]. Although studies have been unable to demonstrate the differences in endothelial cell density, polymegathism and pleomorphism of diabetic patients compared to control groups[14-16,20,21,23-25], some studies in diabetic patients have found a decreased endothelial cell density, increased polymegathism and pleomorphism[14-16,21,23-26]. However, these endothelial changes did not correlate with age, duration of diabetes, stage of diabetic retinopathy, or glycemic control[15,16,21]. Some reports found an increased polymegathism and pleomorphism with increased severity of diabetic retinopathy, and an increased corneal thickness in diabetes[15,20,24,26,27]. Corneal thickness, an indicator of endothelial function, was not increased in some studies[16,28].

The purpose of this study was to analyse the corneal thickness and endothelial cell morphology of Thai patients with diabetes mellitus compared to normal subjects, and to determine the correlation with age, duration of diabetes, stage of diabetic retinopathy and glycemic control.

MATERIAL AND METHOD

Sixty eyes of thirty patients with diabetes mellitus and sixty eyes of thirty healthy nondiabetic subjects as the control group were studied at Siriraj Hospital from July 1999 through April 2000. All subjects had clear corneas and did not have any medical disease that required topical or systemic treatment that had known effects on corneal thickness, endothelial cell density and morphology, except diabetes mellitus. Subjects with a history of contact lens use, glaucoma, intraocular surgery, inflammation or trauma, and use of ocular medications within two weeks of the study were excluded.

A complete ocular examination was performed including visual acuity, slit-lamp biomicroscopy, applanation tonometry and dilated indirect ophthalmoscopy. The diabetic retinopathy was classified into nonproliferative diabetic retinopathy (NPDR), severe nonproliferative diabetic retinopathy (SNPDR) and proliferative diabetic retinopathy (PDR)[29]. The retinal changes in NPDR included microaneurysms, dot and blot intraretinal hemorrhages, retinal edema and hard exudates, and those of SNPDR included dilation and beading of retinal veins, retinal hemorrhage, intraretinal microvascular abnormalities (IRMA) and cottonwool spots. PDR is characterized by extraretinal fibrovascular proliferation extending beyond the internal limiting membrane.

Table 1. Corneal thickness and endothelial cell morphology of diabetic patients compared with control subjects.

	Control		Diabetes		p -value
	mean ± SD	min, max	mean ± SD	min, max	
Thickness (μm)	526.7 ± 32.2	462, 618	534.4 ± 39.5	448, 633	0.249
MCA (μm^2)*	396.7 ± 25.0	341.5, 447.7	384.4 ± 32.6	303.9, 475.2	0.022
Cell density (cells/mm^2)	2,534.7 ± 159.0	2,233.7, 2,927.9	2,619.5 ± 222.4	2,104.5, 3,290.4	0.013
CV (%)**	41.8 ± 5.6	29.4, 57.0	43.2 ± 5.1	32.7, 56.9	0.149
Hexagonal cells (%)	46.0 ± 6.3	30.3, 60.1	44.9 ± 6.1	26.7, 57.7	0.325

* MCA = mean cell area
** CV = coefficient of variation of cell area

Central corneal thickness of each eye was measured with an ultrasonic pachymeter (Humphrey Ultrasonic Pachymeter model 855). Central corneal endothelial photographs were obtained with a contact specular microscope (Tomey endothelial Cell Analyzer EM-1020 version 1.20). A computer-assisted endothelial analysis system calculated the mean and standard deviation (SD) of endothelial cell area, CV, endothelial cell density and percentage of hexagonal endothelial cells. Serum glycosylated hemoglobin levels were collected and analysed.

Each variable in the diabetic group was compared with the control group using a Student *t*-test. Comparisons for sample means among multiple groups were analysed by an analysis of variance (ANOVA). For simultaneous multiple comparisons, a Bonferroni modification of the Student *t*-test was applied. Correlations between variables were evaluated with Pearson and Spearman correlation coefficients (r). Multiple regression analysis evaluated the relative importance of multiple relationships.

RESULTS

The mean age of the normal healthy control subjects and diabetic patients (±SD) was 60.4 ± 11.7 (range 41-82 years) and 60.0 ± 9.1 years (range 40-77 years) respectively, without statistically significant difference in female preponderance (2:1) in both groups.

The mean glycosylated hemoglobin (HbA_{1C}) level (±SD) was 8.5 ± 2.6 per cent (range 5.5-14.3%). Total serum glycosylated hemoglobin levels, with approximate average blood glucose levels during the previous two to three months, were considered relative indicators of diabetic control. Serum glycosylated hemoglobin of less than 7.5 per cent in 14 patients was considered as good glycemic control. The HbA_{1C} level 7.5 per cent or higher in 16 patients was interpreted as poor control. The HbA_{1C} level was not correlated with the duration of diabetes mellitus and stage of diabetic retinopathy. The mean duration of diabetes mellitus (±SD) was 7.6 ± 5.7 years (range 0.5-20 years). The duration of diabetes mellitus correlated with the stage of diabetic retinopathy ($r = 0.430$, $p = 0.001$).

According to severity of diabetic retinopathy, thirty-eight eyes had no retinopathy (NDR), twelve eyes had nonproliferative diabetic retinopathy (NPDR), five eyes had severe nonproliferative diabetic retinopathy (SNPDR), and five eyes had proliferative diabetic retinopathy (PDR).

There was significant decreased mean endothelial cell area ($p = 0.022$) and increased corneal endothelial cell density ($p = 0.013$) in the diabetic patients. The diabetic corneas showed an increased corneal thickness, an increased coefficient of variation of endothelial cell area, and decreased percentage of hexagonal endothelial cells without statistical significance (Table 1).

The duration of diabetes mellitus correlated significantly with corneal thickness ($r = 0.373$, $p = 0.003$), endothelial cell density and coefficient of variation of endothelial cell area, but correlated inversely with mean endothelial cell area and percentage of hexagonal endothelial cells (Table 2).

The stage of diabetic retinopathy was slightly correlated inversely with mean endothelial cell area ($r = -0.389$, $p = 0.002$) but correlated slightly with endothelial cell density ($r = 0.389$, $p = 0.002$). No correlation between these corneal parameters and age or glycemic control was found (Table 2). It is possible that the predictor of corneal thickness, coefficient of variation of endothelial cell area and percentage of hexagonal endothelial cell was the duration of diabetes mellitus. It is also possible that the predictor of mean endothelial cell

Table 2. Correlation between age, HbA_{1C}, duration of diabetes mellitus, stage of diabetic retinopathy, with corneal thickness, and endothelial cell morphology in diabetic patients.

	Age		HbA_{IC}		Duration of diabetes		Stage of diabetic retinopathy	
	r*	p	r*	p	r*	p	r*	p
Thickness (μm)	-0.043	0.744	-0.087	0.509	0.373	0.003	0.122	0.352
MCA (μm^2)	0.193	0.140	0.016	0.905	-0.262	0.043	-0.389	0.002
Cell density (cells/mm^2)	-0.183	0.162	-0.013	0.922	0.266	0.040	0.389	0.002
CV (%)	0.112	0.396	-0.104	0.430	0.281	0.029	-0.153	0.243
Hexagonal cells (%)	-0.044	0.704	-0.004	0.974	-0.256	0.048	-0.034	0.795

* r = correlation coefficient

Table 3. Predictors of corneal thickness and endothelial cell morphology of diabetic patients from multiple regression analysis.

	Predictors	r*	r^2*	p-value
Thickness (μm)	duration	0.373	0.139	0.003
MCA (μm^2)	stage	-0.314	0.099	0.015
Cell density (cells/mm^2)	stage	0.294	0.086	0.023
CV (%)	duration	0.281	0.079	0.029
Hexagonal cells (%)	duration	-0.256	0.066	0.048

* r = correlation coefficient
** r^2 = coefficient of determination

area and endothelial cell density was stage of diabetic retinopathy (Table 3).

In diabetic patients without diabetic retinopathy, the mean cell area was larger than in patients with severe nonproliferative and proliferative diabetic retinopathy, but mean cell density was less than in patients with severe nonproliferative and proliferative diabetic retinopathy (Table 4). The corneal parameters in the NDR group were insignificantly different in the control group.

In good glycemic control the patients were older, had larger mean cell area and less cell density than in poor glycemic control with statistical significance (Table 5). The corneal parameters in the good glycemic control were approximately the same as nondiabetic subjects.

The mean corneal thickness in diabetic patients was increased as the duration of diabetes mellitus increased (Table 6). The group with a duration of diabetes mellitus of more than 10 years had thicker cornea, less mean cell area, more cell density and coefficient of variation, and less hexagonal cells than nondiabetic subjects with statistical significance (p = 0.005, 0.003, 0.027, 0.026, 0.055, respectively).

DISCUSSION

More coefficient of variation of endothelial cell area, less percentage of hexagonal endothelial cells and thicker diabetic cornea were found to be insignificant because of the small sample size and more patients without diabetic retinopathy. Regarding different instruments, these findings are different from other studies which demonstrated statistically significant changes in these corneal parameters[14,16,20,21,24,26,27]. Our patients were also older than those of previous reports[14-16,20-24]. As polymegathism and pleomorphism increase with age, the differences between patients with diabetes mellitus and controls may be less in older subjects [30]. Another possibility is more coefficient of variation of endothelial cell area and lower percentage of hexagonal endothelial cells in non-diabetic group compared with normal healthy groups in former reports[14-16,20].

Our unexpected findings about more corneal endothelial cell density and less mean endothelial cell area in the diabetic group with severe non-proliferative and proliferative diabetic retinopathy compared with control group were the result

Table 4. Corneal parameters in each stage of diabetic retinopathy.

	Mean ± SD		
	No DR (N=38)	NPDR (N=12)	SNPDR + PDR (N=10)
Age (yrs)	59.9 ± 10.7	61.3 ± 2.7	58.6 ± 7.3
Thickness (μm)	532.3 ± 47.0	534.6 ± 23.4	542.0 ± 19.4
MCA (μm^2)	392.7 ± 36.0*	375.4 ± 15.6	364.0 ± 21.7*
Density (cells/mm^2)	2,568.4 ± 245.5*	2,667.8 ± 106.8	2,756.0 ± 166.2*
CV (%)	43.8 ± 4.8	41.7 ± 6.1	43.0 ± 5.0
Hexagonal (%)	45.0 ± 6.6	44.7 ± 6.1	44.7 ± 4.6

* $p < 0.05$

Table 5. Corneal parameters in good glycemic control compared with poor glycemic control.

	HbA_{1C}		
	< 7.5 n = 14	≥ 7.5 n = 16	p-value
Age (yrs)	64.8 ± 7.1	55.8 ± 8.5	<0.001
Thickness (μm)	526.5 ± 30.1	541.2 ± 45.5	0.152
MCA (μm^2)	393.3 ± 31.1	376.7 ± 32.2	0.048
Density (cells/mm^2)	2,557.1 ± 191.3	2,674.2 ± 235.9	0.041
CV (%)	43.0 ± 6.1	43.4 ± 4.1	0.728
Hexagonal (%)	46.3 ± 4.8	43.7 ± 6.9	0.103

Table 6. Comparison of corneal parameters among different duration of diabetes mellitus.

	Duration of diabetes mellitus		
	< 5 yrs (n=10)	5-10 yrs (n=12)	> 10 yrs (n=8)
Age*	54.7 ± 9.6	65.2 ± 8.2	58.9 ± 4.2
Thickness*	516.6 ± 34.1	530.4 ± 32.1	562.4 ± 42.3
MCA	397.2 ± 33.1	381.8 ± 26.4	372.5 ± 36.5
Density	2,533.2 ± 195.6	2,631.2 ± 179.0	2,710.1 ± 279.4
CV (%)	41.5 ± 4.3	43.2 ± 5.1	45.3 ± 5.4
Hexagonal (%)	47.0 ± 4.0	44.7 ± 7.0	42.5 ± 6.3

* $p \leq 0.001$

of the younger age of the patients. These findings are different from many previous studies which demonstrated a decreased endothelial cell density in diabetic patients, or lack of difference in endothelial cell density between diabetic and control groups(14-16,20,21,23-26). This may be caused by a great deal of individual variability in endothelial cell density (14-18), on which the endothelial cell function is not dependent. Therefore, the greater cell density in our diabetic group should not be interpreted as better endothelial cell function.

On multiple regression analysis, the duration of diabetes mellitus is significantly slightly correlated with corneal thickness, polymegathism and pleomorphism which was not noted in most studies(16,21). This finding suggests a cumulative effect of diabetes on the cornea; the longer the duration of diabetes mellitus, the thicker the cornea,

as shown in Table 6. Comparison of corneal thickness among the diabetic groups with a different duration of diabetes mellitus was a significant variation between the groups ($p = 0.001$). The cornea in the duration of the > 10 years group was significantly thicker than the duration < 5 years group ($p = 0.001$), and the duration of the 5-10 years group ($p = 0.002$). No significant difference in corneal thickness was noted between the duration < 5 years group and the duration of the 5-10 years group ($p = 0.638$). But the cornea in the diabetes mellitus with a duration of > 10 years was significantly thicker and with more polymegathism than the non-diabetic corneas ($p = 0.005$, 0.026). This suggests that the corneal thickness measurement by ultrasonic pachymetry which is simple and not time-consuming may be used as a routine pre-operative corneal evaluation for any intraocular surgery in diabetic patients with a duration of more than ten years. Although, insignificantly thicker corneas in diabetic patients compared to normal subjects, requires a large sample size and further investigation. However, epithelial disease can affect corneal thickness and is a common problem in diabetics. Therefore, some of the changes in corneal thickness in diabetics could be due to epithelial disease.

The stage of diabetic retinopathy is significantly correlated with mean endothelial cell density and inversely with mean endothelial cell area. This might be due to greater endothelial cell density and less mean endothelial cell area in severe non-proliferative and proliferative diabetic retinopathy compared with no diabetic retinopathy and non-diabetic patients. Although the patients without diabetic retinopathy were younger than non-diabetic patients with slightly thicker cornea, more polymegathism and pleomorphism, we did not find a correlation of the investigated corneal parameters with serum glycosylated hemoglobin levels, as in other studies (15,16,21). Because of the older age of the good glycemic control group compared with the poor glycemic control group and the serum glycosylated hemoglobin levels only approximate blood glucose levels during the previous two to three months did not represent the developed disease. Furthermore, good glycemic controls were older than the non-diabetic group with a significance level of $p = 0.031$, with the same thickness and pleomorphism. This finding should be further investigated.

On clinical observation, a normal looking cornea may decompensate following intraocular surgery, whereas another cornea that demonstrated substantial endothelial morphological changes may not decompensate. Morphological analysis of the corneal endothelium alone may not be a sufficient indicator of its functional capacity(31). However, this study evaluated only corneal thickness and endothelial cell morphology. The functional capacity and ultrastructural change of the corneal endothelium of the diabetic patients were not examined. Several studies have demonstrated an increased corneal autofluorescence and endothelial permeability to fluorescein(15,16,24,28,32). Kim et al reported marked irregular F-actin fibers of the corneal endothelium of diabetes which is a major component of the cellular cytoskeleton with importance in maintaining cell shape and barrier function of the corneal endothelium(33).

Changes in corneal endothelial structure alone do not necessarily contribute to alter endothelial function or increase permeability. Young contact lens wearers also have structural endothelial changes without a change in permeability(34). Several studies have evaluated the corneal endothelial functional reserve by contact lens induced hypoxic edema, and found a decreased ability to recover from corneal edema in diabetic corneas, which indicates insufficient reserve to handle the stress of induced corneal edema despite the normal endothelial function in the unstressed resting state(35,36).

The factors causing these structural and functional changes in the diabetic endothelium are not known. Most authors postulate that these changes are due to altered corneal glusidic metabolism leading to sorbitol accumulation(14,16,33,35,37,38). Aldose reductase (AR), the first enzyme of the polyol pathway, has been implicated in the pathogenesis of a number of diabetic complications. Akagi et al demonstrated AR in the human corneal endothelium, so it is possible that AR is involved in the etiology of the endothelial changes in diabetic cornea(39). Ohguro and others demonstrated an improvement of endothelial pleomorphism and polymegathism within three months after treating the human diabetic corneas with topical aldose reductase inhibitor, suggesting that AR may be involved in the etiology of corneal endothelial changes in diabetes(38).

In summary, various clinical observations of abnormalities in diabetic cornea are not unexpected. More subjects and more extensive study are required to confirm these corneal changes and correlations between a number of variables. We suggest that diabetic patients of more than ten years duration should be pre-operatively measured for corneal thickness and specular microscopy to detect endothelial dysfunction. This would serve to remind us to avoid the risk of further corneal endothelial damage to improve the surgical outcome.

ACKNOWLEDGMENT

The authors wish to thank all the technicians, residents and staff in the Department of Ophthalmology, Faculty of Medicine Siriraj Hospital for their great cooperation, and Mr. Suthipol Udompanthurat for his advice in statistics.

(Received for publication on February 5, 2001)

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ความผิดปกติของกระจกตาในผู้ป่วยเบาหวาน

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เพื่อศึกษาความหนาของกระจกตาและลักษณะเซลล์เอ็นโดธิเลี่ยมของกระจกตา ในผู้ป่วยเบาหวาน 30 ราย (60 ตา) เปรียบเทียบกับคนปกติ 30 ราย (60 ตา) และศึกษาว่าลักษณะดังกล่าวมีความสัมพันธ์กับระยะเวลาที่เป็นเบาหวาน ความรุนแรงของจอประสาทตาเสื่อมจากเบาหวานและการควบคุมเบาหวานหรือไม่ จากการตรวจวัดความหนากระจกตาด้วยคลื่นเสียง (ultrasonic pachymeter) ดูลักษณะเซลล์เอ็นโดธิเลี่ยมด้วยกล้องขยายสเปคคิวล่า (Specular microscope) พบว่าในผู้ป่วยเบาหวานมีปริมาณเซลล์เอ็นโดธิเลี่ยมมากกว่าคนปกติ แต่มีขนาดเซลล์แปรปรวนมากกว่า ปริมาณเซลล์รูปร่างหกเหลี่ยมมีน้อยกว่า และกระจกตาหนามากกว่าคนปกติเล็กน้อยอย่างไม่มีนัยสำคัญทางสถิติ ระยะเวลาที่เป็นเบาหวานมีความสัมพันธ์กับรูปร่างของเซลล์ ขนาดเซลล์ที่แปรปรวนและความหนากระจกตาอย่างมีนัยสำคัญทางสถิติ แต่ความรุนแรงของจอประสาทตาเสื่อมจากเบาหวานมีความสัมพันธ์กับปริมาณเซลล์ และการเปลี่ยนแปลงของกระจกตาไม่สัมพันธ์กับการควบคุมเบาหวาน กระจกตาในผู้ป่วยเบาหวานจะมีความหนามากกว่า ตามระยะเวลาที่เป็นเบาหวานนานเกิน 10 ปี จึงสมควรตรวจวัดความหนาของกระจกตาในผู้ป่วยเบาหวานที่เป็นมานานก่อนทำผ่าตัดตา เพื่อระมัดระวังความเสี่ยงของกระจกตาเสื่อมภายหลัง

คำสำคัญ : ปริมาณเซลล์เอ็นโดธิเลี่ยม, ขนาดเซลล์ที่แปรปรวน, รูปร่างเซลล์หลายแบบ, ความหนากระจกตา, โรคเบาหวาน

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จดหมายเหตุทางแพทย์ ฯ 2544; 84: 1075–1083

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