Section: Original Research

Original Research Article

CORNEAL ENDOTHELIAL ALTERATIONS IN TYPE 2 DIABETES MELLITUS: A COMPARATIVE SPECULAR MICROSCOPY STUDY

Aditya Singh Baghel[1], Suman Chahar[2], Manish Kumar Prajapat[3], T.S. Ahluwalia[4]

[1]*Junior Resident, Department of Ophthalmology, National Institute of Medical Sciences & Research (NIMS), Jaipur, Rajasthan, India*
[2]*Assistant Professor, Department of Ophthalmology, National Institute of Medical Sciences & Research (NIMS), Jaipur, Rajasthan, India*
[3]*Associate Professor, Department of Ophthalmology, National Institute of Medical Sciences & Research (NIMS), Jaipur, Rajasthan, India*
[4]*Professor, Department of Ophthalmology, National Institute of Medical Sciences & Research (NIMS), Jaipur, Rajasthan, India*

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Corresponding Author:
Dr. Aditya Singh Baghel,
Junior Resident, Department of Ophthalmology, National Institute of Medical Sciences & Research (NIMS), Jaipur, Rajasthan, India.
Email: dr.aditya.s.baghel@gmail.com

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) has been associated with structural and functional changes in the corneal endothelium. Non-contact specular microscopy allows quantitative assessment of endothelial cell density (ECD), coefficient of variation (CV), hexagonality (HEX) and central corneal thickness (CCT). This study compared these parameters in adults with T2DM and non-diabetic controls.

Materials and Methods: This comparative cross-sectional study was conducted in the Departments of Ophthalmology and Endocrinology, National Institute of Medical Sciences and Research (NIMS&R), Jaipur, over 18 months. One hundred and fifty participants aged ≥40 years were enrolled (75 with T2DM and 75 non-diabetic controls). After a complete ophthalmic examination, non-contact specular microscopy was performed with a Nidek CEM-530. ECD, CV, HEX and CCT were compared between groups, and correlations with diabetes duration and HbA1c were examined.

Results: Mean ECD was significantly lower in T2DM than in non-diabetic participants in the right eye (2470.23 ± 131.08 vs 2662.81 ± 175.58 cells/mm²; $p<0.001$) and the left eye (2469.32 ± 137.83 vs 2661.01 ± 169.73 cells/mm²; $p<0.001$). CV was significantly higher in participants with T2DM in both eyes (right: 37.36 ± 2.81% vs 30.20 ± 1.21%; left: 37.91 ± 2.38% vs 29.81 ± 1.15%; both $p<0.001$). CCT was significantly greater in T2DM (right: 514.04 ± 16.59 vs 504.01 ± 6.08 µm; left: 515.17 ± 18.07 vs 505.23 ± 6.26 µm; both $p<0.001$). HEX did not differ significantly between groups (right eye $p=0.527$; left eye $p=0.418$). Duration of diabetes showed a weak positive correlation with CCT ($r=0.26$, $p=0.024$).

Conclusion: T2DM was associated with lower ECD, greater polymegathism and greater CCT, without a significant difference in hexagonality. Specular microscopy can detect subclinical endothelial change in patients with T2DM and may be useful when endothelial reserve is relevant, including before intraocular surgery.

Keywords: Type 2 diabetes mellitus; corneal endothelium; specular microscopy; endothelial cell density; coefficient of variation; hexagonality; polymegathism; central corneal thickness.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disease defined by persistent hyperglycaemia and by long-term microvascular and systemic complications.[1,2] Diabetic retinopathy is the best recognised ocular complication, but hyperglycaemia can affect almost every ocular tissue, including the cornea. Diabetic keratopathy may include reduced corneal sensitivity, delayed epithelial healing, altered stromal physiology and impaired endothelial integrity.[3] These changes may be clinically subtle at first, yet they are relevant to corneal health and visual function.[3,4]

The corneal endothelium is a monolayer lining the posterior cornea and maintains transparency by regulating stromal hydration.[5] Human corneal endothelial cells have very limited mitotic capacity in vivo; cell loss is therefore compensated mainly by enlargement and change in shape of remaining cells. Three parameters commonly used to describe endothelial health are endothelial cell density (ECD), coefficient of variation of cell area (CV) and the percentage of hexagonal cells (HEX).[6] An increase in CV (polymegathism) and a decrease in HEX (pleomorphism) suggest endothelial stress or reduced reserve.[7]

Chronic hyperglycaemia and the associated metabolic milieu can injure the corneal endothelium. Larger mean cell area, lower HEX, lower ECD and other morphological changes have been reported in diabetes.[8] Impaired endothelial pump function may increase stromal hydration and thereby increase central corneal thickness (CCT). Assessment of the endothelium may therefore identify diabetic ocular involvement before overt corneal disease is visible at the slit lamp.[9,10]

Specular microscopy is a non-invasive method for imaging and quantifying the endothelial mosaic in vivo. It yields ECD, mean cell area, CV, HEX and CCT.[3,6] These measurements allow an objective estimate of endothelial morphology and functional reserve, which is particularly relevant in patients who may later need intraocular surgery, when adequate reserve is required to maintain corneal clarity.[7]

Published estimates of the diabetic effect on the endothelium are not uniform. A systematic review and meta-analysis of 14 studies found lower ECD and HEX in diabetes, together with higher mean cell area, CV and CCT.[1] A separate systematic review focused on pachymetry likewise reported greater CCT in diabetes.[11] Experimental work has linked hyperglycaemia to impaired endothelial mitophagy and related metabolic injury.[12]

Findings specific to type 2 diabetes mellitus are also mixed. Some studies have shown lower ECD and higher CCT in T2DM, whereas others have found little or no morphological difference.[2,6,13] The size of the effect may depend on duration of diabetes, glycaemic control, age, retinopathy severity, sample size and the instrument used.[5,14]

Because early endothelial change may precede clinically obvious keratopathy, a standardised non-contact comparison of diabetic and non-diabetic eyes can provide baseline data for ophthalmic assessment and surgical planning.[12-15]

The aim of this study was to compare ECD, CV, HEX and CCT, measured with a Nidek CEM-530 non-contact specular microscope, in patients with T2DM and in non-diabetic controls, and to examine associations with duration of diabetes and HbA1c.

MATERIALS AND METHODS

Research Design and Setting: A comparative cross-sectional study was conducted in the Department of Ophthalmology, in collaboration with the Department of Endocrinology, NIMS&R, Jaipur, over 18 months. The study began after approval from the Institutional Scientific and Ethics Committee (approval number to be inserted). All participants provided written informed consent before enrolment. The study was not prospectively registered.

Study Population and Sample Size: One hundred and fifty adults aged ≥40 years were enrolled: 75 with type 2 diabetes mellitus (Group A) and 75 without diabetes (Group B). Sample size was calculated for a comparison of two independent means with equal allocation, taking endothelial cell density (ECD) as the primary outcome. Using the pooled mean difference of 114.29 cells/mm² reported by Sharifi et al. and an assumed common standard deviation of 250 cells/mm², a two-sided α of 0.05 and 80% power required 75 participants in each group [$n = 2(Z_{1-\alpha/2} + Z_{1-\beta})^2\sigma^2/\delta^2$]. A sensitivity calculation based on Taşlı et al. (mean ECD difference 187.62 cells/mm²; pooled SD approximately 311 cells/mm²) required 44 participants per group; the enrolled size therefore also covered that estimate. Participants were recruited by convenience sampling from the ophthalmology and endocrinology outpatient clinics.

T2DM was confirmed from clinical records using American Diabetes Association diagnostic criteria. The non-diabetic group comprised individuals with no history of diabetes or other major systemic disease who attended the ophthalmology outpatient department for unrelated ocular complaints and whose recorded fasting plasma glucose (<100 mg/dL) and HbA1c (<5.7%) were in the non-diabetic range.

Exclusion criteria were corneal opacity or dystrophy (including clinically evident guttata), active uveitis, previous ocular trauma or intraocular surgery, current or recent contact-lens wear, and any media opacity that prevented adequate specular imaging.

Clinical and Ophthalmologic Evaluation: Demographic data, duration of diabetes, antidiabetic treatment and ocular history were recorded on a structured proforma, together with fasting plasma glucose and HbA1c. All participants underwent uncorrected and best-corrected visual acuity (decimal notation), slit-lamp biomicroscopy, Goldmann applanation tonometry and dilated fundus examination. Diabetic retinopathy, when present, was graded using a standard clinical classification.

Specular Microscopy: Non-contact specular microscopy of the central corneal endothelium was performed on both eyes with a Nidek CEM-530 under standardised conditions, before pupillary dilatation. The parameters recorded were endothelial cell density (ECD, cells/mm²), coefficient of variation of cell area (CV, %), percentage of hexagonal cells (HEX, %) and central corneal thickness (CCT, µm). Three analysable central images were obtained for each eye; frames with poor focus or incomplete mosaics were discarded and the mean of the three accepted readings was used for analysis. The instrument's automated centre analysis was used, and the number of cells counted per image was recorded.

Statistical Analysis: Data were analysed with SPSS version 25. Continuous variables are presented as mean ± standard deviation and categorical variables as number (percentage). Between-group comparisons used Student's t-test or the Mann–Whitney U test, according to distribution. Associations of ECD, CV, HEX and CCT with duration of diabetes and HbA1c were examined with Pearson correlation. A two-sided p-value <0.05 was considered statistically significant. Right and left eyes were analysed separately; inter-eye correlation was not modelled and is acknowledged as a limitation.

RESULTS

One hundred and fifty participants were enrolled (75 with T2DM and 75 non-diabetic controls). Baseline demographic and glycaemic characteristics are shown in [Table 1]. Age and sex were comparable between groups. Fasting plasma glucose and HbA1c were significantly higher in the T2DM group (both p<0.001). Mean duration of diabetes was 11.11 ± 4.62 years. [Table 2] shows that best-corrected visual acuity (decimal) was lower and intraocular pressure higher in both eyes of participants with T2DM than in controls. Occupational categories were recorded for description only and were not used in the endothelial analyses.

Corneal endothelial parameters are summarised in [Table 3]. Participants with T2DM had significantly lower ECD and significantly higher CV and CCT in both eyes; HEX did not differ significantly between groups. Fundus findings are shown in [Table 4]. Diabetic retinopathy was present in 34.7% of the T2DM group and was almost entirely mild non-proliferative diabetic retinopathy (NPDR). [Table 5] shows right-eye correlations of diabetes-related variables with endothelial parameters. Duration of diabetes had a weak positive correlation with CCT (r=0.26, p=0.024) and was not significantly correlated with ECD, CV or HEX. HbA1c was not significantly correlated with any endothelial parameter.

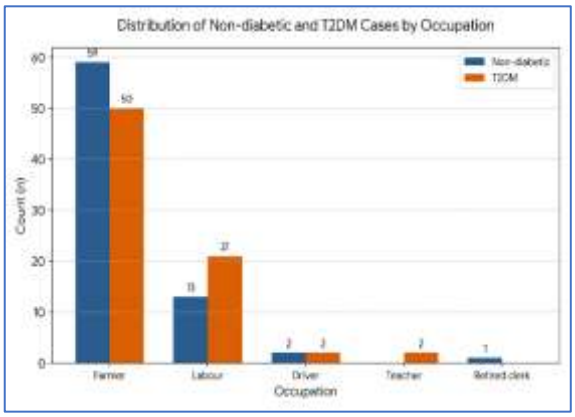

Figure 1 omitted: occupational distribution was descriptive only and was not retained.

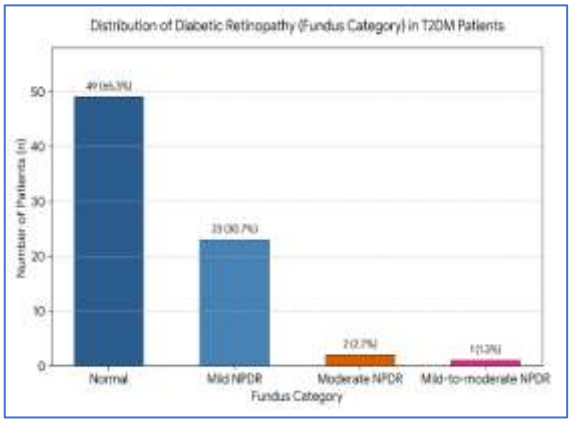

Figure 2 omitted: retinopathy grades are fully reported in Table 4.

Table 1: Baseline Demographic and Glycaemic Characteristics

Variable	T2DM (n=75)	Non-diabetic (n=75)	Between-group difference	p-value
Age, years, mean ± SD	65.68 ± 9.33	62.77 ± 9.86	2.91 (95% CI −0.19 to 6.01)	0.066
Male, n (%)	38 (50.7)	39 (52.0)	—	1.000
Female, n (%)	37 (49.3)	36 (48.0)	—	1.000
Duration of T2DM, years	11.11 ± 4.62	—	—	—
Fasting blood glucose, mg/dL	113.01 ± 20.58	98.79 ± 8.62	14.22 (95% CI 9.11–19.33)	<0.001
HbA1c, %	7.95 ± 0.83	5.21 ± 0.28	2.74 (95% CI 2.54–2.94)	<0.001

Table 2: Comparative Ophthalmic Parameters

Parameter	T2DM	Non-diabetic	Mean difference (95% CI)	p-value
UCVA, right eye	0.09 ± 0.05	0.10 ± 0.08	−0.01 (−0.03 to 0.01)	0.238
UCVA, left eye	0.10 ± 0.07	0.09 ± 0.06	0.01 (−0.01 to 0.03)	0.674
BCVA, right eye	0.15 ± 0.08	0.23 ± 0.14	−0.08 (−0.12 to −0.04)	<0.001
BCVA, left eye	0.16 ± 0.09	0.22 ± 0.12	−0.06 (−0.09 to −0.03)	0.001
IOP, right eye, mmHg	17.71 ± 2.12	14.81 ± 1.33	2.90 (2.33 to 3.47)	<0.001
IOP, left eye, mmHg	17.61 ± 2.07	14.55 ± 1.02	3.06 (2.53 to 3.59)	<0.001

Table 3: Comparison of Corneal Endothelial Parameters Between Study Groups

Parameter	T2DM	Non-diabetic	Mean difference (95% CI)	p-value	Cohen's d
ECD, right eye (cells/mm²)	2470.23 ± 131.08	2662.81 ± 175.58	−192.58 (−242.61 to −142.55)	<0.001	−1.24
ECD, left eye (cells/mm²)	2469.32 ± 137.83	2661.01 ± 169.73	−191.69 (−241.60 to −141.78)	<0.001	−1.24
CV, right eye (%)	37.36 ± 2.81	30.20 ± 1.21	7.16 (6.46–7.86)	<0.001	3.31
CV, left eye (%)	37.91 ± 2.38	29.81 ± 1.15	8.10 (7.49–8.71)	<0.001	4.33
HEX, right eye (%)	51.01 ± 3.69	50.65 ± 3.24	0.36 (−0.76 to 1.48)	0.527	0.10

HEX, left eye (%)	50.29 ± 4.15	50.77 ± 2.99	−0.48 (−1.65 to 0.69)	0.418	−0.13
CCT, right eye (µm)	514.04 ± 16.59	504.01 ± 6.08	10.03 (5.98–14.08)	<0.001	0.80
CCT, left eye (µm)	515.17 ± 18.07	505.23 ± 6.26	9.94 (5.55–14.33)	<0.001	0.74

Table 4: Fundus Status and Diabetic Retinopathy in the Study Population

Fundus status	T2DM, n (%)	Non-diabetic, n (%)
Normal fundus	49 (65.3)	75 (100)
Any NPDR	26 (34.7)	0 (0.0)
Mild NPDR	23 (30.7)	0 (0.0)
Moderate NPDR	2 (2.7)	0 (0.0)
Mild-to-moderate NPDR	1 (1.3)	0 (0.0)

Table 5: Correlation of Diabetes-Related Variables With Right-Eye Corneal Endothelial Parameters

Predictor	Corneal parameter	Pearson's r	p-value	Interpretation
Duration of diabetes	ECD	−0.11	0.336	Not significant
Duration of diabetes	CV	−0.07	0.565	Not significant
Duration of diabetes	HEX	−0.05	0.690	Not significant
Duration of diabetes	CCT	+0.26	0.024	Significant weak positive correlation
HbA1c	ECD	−0.09	0.427	Not significant
HbA1c	CV	+0.02	0.846	Not significant
HbA1c	HEX	+0.01	0.918	Not significant
HbA1c	CCT	+0.13	0.252	Not significant

DISCUSSION

In this comparative study, corneal endothelial parameters differed between patients with T2DM and non-diabetic controls. ECD was lower, and CV and CCT were higher, in both eyes of participants with T2DM; HEX did not differ significantly. These findings support subclinical endothelial involvement in T2DM even when the cornea appears quiet on routine slit-lamp examination.

Endothelial Cell Density: Both eyes of patients with T2DM showed lower ECD, with a mean between-group difference of about 192 cells/mm². This direction of effect is consistent with the meta-analysis by Sharifi et al,[1] who reported significantly lower ECD in diabetes. Taşlı et al,[2] likewise found lower ECD in T2DM (2427.94 ± 427.10 vs 2615.56 ± 106.80 cells/mm² in controls). Kim et al,[4] and Jha et al,[5] also reported reduced ECD in diabetic eyes.

Opala et al,[9] using the same Nidek CEM-530 device, found lower ECD even in patients with well-controlled T2DM. Agreement across cohorts and instruments suggests that endothelial cell loss is a recurrent response to the diabetic milieu rather than an artefact of a single device or population. Age remains an important determinant of ECD; our T2DM group was nearly three years older than the control group (p=0.066), so part of the observed difference could be age-related. In the absence of an age-adjusted model (for example ANCOVA), the diabetic effect on ECD should be interpreted with that caveat.

Endothelial Cell Morphology: CV was significantly higher in the T2DM group, indicating greater polymegathism. This finding agrees with Sharifi et al,[1] Taşlı et al,[2] and Kim et al.[4]

HEX, by contrast, did not differ between groups. That result diverges from Sharifi et al,[1] Taşlı et al,[2] Jha et al,[5] and Firdous et al,[10] who reported lower hexagonality in diabetes, but it is consistent with Beato et al,[6] and Opala et al,[9] who found no clear hexagonal difference. Discrepancies may reflect differences in diabetes duration, glycaemic control, age, retinopathy severity, sample characteristics and specular analysis settings. Mean HEX was close to 50% in both of our groups, which is lower than values reported in some CEM-530 series. Replication should specify analysis mode and the number of cells counted per image, as we have now done in Methods. Higher CV with preserved HEX in the same cohort suggests that polymegathism and measurable cell loss need not be accompanied by a detectable fall in hexagonality at every disease stage. Experimental studies offer a plausible mechanism: hyperglycaemia can impair mitochondrial quality control in endothelial cells through reduced mitophagy, greater susceptibility to oxidative injury and altered glutamine metabolism.[12-14] Those data are laboratory-based and should be read as biological context rather than as a direct explanation of the present measurements.

Central Corneal Thickness: Mean CCT was approximately 10 µm greater in both eyes of participants with T2DM. That increment is close to the pooled CCT difference reported by Sharifi et al,[1] Taşlı et al,[2] and Kim et al,[4] described similar thickening; Jha et al,[5] reported a larger increase. Greater CCT is consistent with reduced pump function and increased stromal hydration. Goldmann-applanation intraocular pressure was also higher in the T2DM group. Because applanation readings rise with CCT, part of the IOP difference may be pachymetric. The observed CCT increment of about 10 µm is nevertheless too small to account fully for a mean IOP difference of about 3 mmHg.

Duration of diabetes showed a weak positive correlation with CCT (r = 0.26, p = 0.024), in keeping with Kim et al,[4] who identified longer duration as a factor associated with endothelial and pachymetric change. Duration was not significantly associated with ECD, CV or HEX in this sample.

Glycaemic Control and Diabetic Retinopathy: A single HbA1c value was not significantly associated with ECD, CV, HEX or CCT. Kim et al,[4] likewise found a limited relationship with a contemporaneous HbA1c, whereas Chowdhury et al,[3] reported associations with ECD, CV and CCT. One clinic HbA1c cannot capture cumulative glycaemic exposure, so the absence of correlation in the present data should not be taken as evidence that long-term control is irrelevant to the endothelium.

Diabetic retinopathy was present in 34.7% of the T2DM group, almost all of it mild NPDR. Categories were grouped as none, mild NPDR and moderate NPDR; mixed labels such as "mild-to-moderate" were avoided in interpretation. Previous work has linked more advanced retinopathy with greater endothelial abnormality.[2,5,8] The present sample contained too few eyes with moderate or worse NPDR for a robust severity analysis. Endothelial change and retinopathy may both reflect microvascular burden, but this study cannot establish that sequence.

Clinical Implications: Taken together, T2DM was associated with lower ECD, greater polymegathism and greater CCT. That pattern implies reduced endothelial reserve and is relevant when intraocular surgery is planned. Specular microscopy adds quantitative information to the slit-lamp examination, particularly in patients with long-standing diabetes. This study did not assess postoperative corneal outcomes, so the surgical implication remains inferential.

Limitations: This study is limited by its cross-sectional, single-centre design, modest sample size and convenience sampling. Age was not adjusted despite a near-significant age difference. Smoking, hypertension, dyslipidaemia, renal disease and details of diabetes treatment were not modelled. Only one HbA1c value was available. Both eyes were analysed separately without a clustered or mixed-effects model, which can narrow confidence intervals. Visual acuity was recorded in decimal notation; future reports should also consider logMAR. Control participants were recruited from an ophthalmology clinic rather than from a population sample, which may limit generalisability. Inter-observer repeatability of CEM-530 traces was not assessed. Control-group SDs for CV and CCT were unusually small; the raw image set should be retained so that this distribution can be verified.

Overall Interpretation: These data add to the evidence that T2DM is associated with measurable subclinical change in the corneal endothelium. In this cohort, ECD, CV and CCT were more sensitive to the diabetic grouping than HEX. Larger, longitudinal, multicentre studies with age adjustment, inter-eye statistical modelling and richer metabolic and microvascular data are needed before corneal endothelial indices can be proposed as early markers of systemic or ocular diabetic complications.

CONCLUSION

Patients with T2DM had significantly lower endothelial cell density, higher coefficient of variation and greater central corneal thickness in both eyes than non-diabetic controls, without a significant difference in hexagonality. The pattern is consistent with endothelial cell loss, polymegathism and altered corneal hydration.

These quantitative abnormalities were present in corneas that appeared normal on slit-lamp examination. Duration of diabetes showed only a weak correlation with CCT; none of the endothelial parameters correlated significantly with a single HbA1c value.

Non-contact specular microscopy is a practical way to detect early endothelial change in T2DM and may help identify reduced endothelial reserve when intraocular surgery is being considered. Larger multicentre longitudinal studies that adjust for age and other systemic confounders are needed to define the prognostic value of these changes.

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