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Original Research Article

Comparison of endothelial cell loss using specular microscopy in diabetic verses non-diabetic patients undergoing phacoemulsification surgery at an eye hospital in central India

Prerna Upadhyaya[1], Aishwarya Singh Raghuvanshi[1]*

[1] *Dept. of Ophthalmology, Sewa Sadan Eye Hospital, Bhopal, Madhya Pradesh, India*

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ABSTRACT

Background: Diabetes mellitus causes several ophthalmic complications including retinopathy, cataract, and damage to the anterior chamber of the eye including various layers of the cornea.
Aim: To compare endothelial cell loss after phacoemulsification surgery for cataracts among diabetics and non-diabetic patients.
Materials and Methods: A single-centre, hospital-based, prospective, comparative, 1:1 observational study enrolling participants a total of 200 participants were enrolled (100 diabetic and non-diabetic) and were followed up from the preoperative period to 3 months after surgery. The data pertaining to endothelial cell density, coefficient of variance, endothelial cell hexagonality and central corneal thickness were recorded using specular microscopy before, 1-, 7-, 30-, and 90 days after cataract surgery.
Results: There was a significant difference ($p<0.05$) in the endothelial cell density, coefficient of variance, endothelial cell hexagonality, and central corneal thickness among the diabetic and non-diabetic patients at each follow-up visit after surgery. The ECD was comparatively lowest among the participants with >10 years of diabetes and highest among patients with < 5 years of diabetes. The percentage change in ECD on days 1-, 7-, 30-, and 90 days of cataract surgery among the diabetic patients was 8.8%, 11.7%, 12.9% and 18.7%, respectively. The mean ECH among the patients with < 5 years, 5-10 years, and > 10 years of diabetes was 65.7%, 62.5% and 57.5%, respectively.
Conclusion: In comparison to non-diabetic patients, there was a considerable loss of endothelial cell function among diabetic individuals following cataract surgery. Furthermore, a slower and poorer healing response was seen in patients with diabetes.

1. Introduction

Together with the sclera, the cornea is a clear avascular tissue that forms the outer section of the eye.[1] The cornea's primary source of nutrition is the aqueous humour. Blood flow is provided via small capillaries at the cornea's periphery as well as end branches of the face and ophthalmic arteries via aqueous humour and the tear film.[2] The corneal endothelium has the lowest mitotic activity of all cell layers. Along the lateral boundaries, adjacent cells share significant lateral interdigitations and have both gap and tight connections.[3] Damage to the endothelium is potentially more severe than damage to the other corneal layers, given the importance of its function, as it can result in cell death and irreversible damage to the endothelial cytoskeleton, ultimately compromising visual function.[4–6] Adult endothelial cells do not replicate and regenerate.[6] Corneal Endothelium Cells (CEC) lack a considerable ability for in vivo regeneration, preventing

* *Corresponding author.*
E-mail address: aishwaryasr1702@gmail.com (A. S. Raghuvanshi).

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them from replacing injured or dead cells because CECs are arrested in the G1 phase of the cell cycle.[4] To maintain proper structure and function, endothelial cells respond to minor damage by stretching and migrating centripetally into the injured area; however, an increase in cell size (polymegathism) and variation in cell shape (pleomorphism) correlates to a decrease in the cells' ability to hydrate the cornea.[1] Throughout adulthood, normal corneal endothelial cell density falls by an average of 0.6% every year with a progressive increase in polymegathism and pleomorphism.[7] The corneal endothelium is essential for the maintenance of normal corneal hydration, thickness, and transparency.[8] Loss or damage of endothelial cells leads to an increase in corneal thickness (oedema) which may ultimately induce corneal decompensation and loss of vision. Corneal blindness represents the fourth leading cause of blindness worldwide (5.1%) and is a major cause of visual impairment after cataracts, glaucoma, and age-related macular degeneration.[9] Endothelial cell density can be significantly decreased as a result of trauma, refractive surgery, previous penetrating, or endothelial keratoplasty or stress caused by disorders such as diabetes, glaucoma, or endothelial dystrophies.

Diabetes Mellitus (DM) is arguably the most significant non-infectious pandemic of the current millennium. Worldwide, cataracts are the main cause of blindness. The pathophysiology of diabetic cataracts is associated with chronic hyperglycaemia increasing reactive oxygen species.[10,11] Patients with diabetes acquire cataracts at a younger age than those without diabetes. Even though cataract surgery can restore their eyesight, diabetic patients may be more prone to corneal problems after the procedure, according to several recent research.[10,11] Patients with diabetes who undergo cataract surgery are more likely to exhibit intraocular inflammation.[12] The process by which diabetes damages corneal endothelial cells is not well understood, however numerous theories have been put forward. In diabetic patients, hyperglycaemia induces metabolic stress, which can result in decreased endothelial cell density and increased pleomorphism and polymegathism.[13] Due to the accumulation of advanced glycation end products in ocular endothelial cells, diabetes can cause oxidative stress.[14]

The most frequent surgical therapy for treating cataracts, phacoemulsification, can enhance visual acuity with few risks. Although phacoemulsification's safety has been significantly established in recent years, surgeons should be cognizant of the potential risk factors associated with more difficult eyes.[15–17] Despite the possible benefits of phacoemulsification, corneal endothelial cell death can occur because of ultrasonic energy injury during surgery. Hence, after phacoemulsification, ocular endothelial cell destruction remains a major problem.[17–19] Previous research has yielded conflicting findings regarding whether diabetic patients have an increased risk of ECD loss following phacoemulsification. Diabetes-related endothelial cell loss (ECL) during phacoemulsification cataract surgery has been the subject of inconsistent findings in previous research.[12,17,20–23] Similarly, various researchers have examined the effect of cataract surgery aggressiveness on the endothelial cell numbers and morphology of diabetic patients, but none have reached definitive conclusions. Hence, the present study aimed to measure, evaluate, and compare the endothelium morphology among diabetic and non-diabetic patients before and after phacoemulsification for cataracts. More specifically, this study was designed to assess the change in endothelial cell density, endothelial cell area, coefficient of variation (CV), and hexagonality between diabetes and non-diabetic patients.

1.1. *Research question*

What is the pattern of changes in endothelial cell morphology after the phacoemulsification procedure for cataracts among diabetic and non-diabetic participants?

2. Materials and Methods

1. This was a single-centre, in-patient, hospital-based, comparative, prospective, observational study.
2. Department of Ophthalmology, Sewa Sadan Eye Hospital, Bhopal, Madhya Pradesh. It is a state of art institute dedicated to ophthalmology.
3. 9 months; from April 2022 to December 2022 divided into the following phases.

The following primary outcome(s) were measured.

1. Endothelial cell density
2. iCoefficient of variation of endothelial cell
3. Endothelial cell hexagonality
4. Central cornea thickness

Corneal endothelial morphological properties were assessed using the non-contact specular microscope (EM-4000). All the measurements were taken by the principal investigator to avoid inter-observer variation.

All the primary outcomes were measured pre-operatively, at postoperative days 1, 7, 30, and 90 days after surgery.

The study was terminated if:

1. A participant decided to withdraw from the study,
2. If surgery was postponed for any reason,
3. A participant did not report for follow-up,
4. After completion of the data collection.

2.1. *Comparative groups*

The primary outcomes were measured and compared between the following two groups:

1. Group D: Patient having diabetes
2. Group N: Patients without diabetes

2.2. Inclusion criteria

1. Participants were diagnosed to have senile cataracts only.
2. NS Grad II or III cataract.
3. Participants aged between 50-80 years (both inclusive).
4. Participants of all genders.
5. Patients consenting to participate in the study.

2.3. Exclusion criteria

1. Any other type of cataract other than age-related
2. Pre-operative endothelial cell count < 1500 cells/mm2
3. Pupillary dilation< 7mm
4. Intra-operative complication
5. Corneal diseases
6. Other causes of decreased vision (diabetic retinopathy, glaucoma)
7. A patient refused to take part in the study.

2.4. Participants definition

A diabetic or non-diabetic patient undergoing elective cataract surgery (phacoemulsification), fulfilling the above-mentioned selection criteria and consented to participate in the present study.

2.5. Sample size

The minimum required sample size for the study was calculated using the formula recommended by Zhong B (2019)[24]. The minimum sample size was calculated as 200 participants: with 100 participants in each of the two groups. All participants who fulfilled the selection criteria were recruited into the present study until the desired sample size in each group was completed.

2.6. Informed consent

Everyone who participated was provided with a copy of the consent form to read. Following that, the contents of the consent form were explained to every potential participant in easy-to-understand language. All of the participants' inquiries about the study, treatment, investigations, and the confidentiality of their data were addressed and answered. It was made clear to the participants, both verbally and in writing, that they are free to discontinue their participation in the research at any moment. After that, those participants who were willing to take part were asked to sign the consent form.

2.7. Data collection

The data were collected in a paper-based questionnaire. The questionnaire had 4 parts as follows:

1. Part 1: Demographic, anthropometric and clinical history.
2. Part 2: Preoperative and Intraoperative details.
3. Part 3: Post-operative details.
4. Part 4: Follow-up details

2.8. Plan and procedure

1. The surgical team completed a detailed history and a thorough clinical examination of every patient. Appropriate laboratory and radiological investigations were conducted. A detailed pre-anaesthetic evaluation was completed one day before the surgery by the anaesthesiologist's team.
2. The examination included best corrective visual acuity, slit lamp examination, fundus examination & Goldman applanation tonometry. Lens opacities classification system-III (LOCS-III) was used to grade the cataract.
3. After the screening, informed consent was obtained from the subjects. The lead investigators approached all prospective volunteers and provided them with a thorough explanation of the study's methodology and its roles (and implications). The proper protocol was followed to obtain participants' written informed consent.
4. On arrival in the operating room, the identity of the participant and the consent were verified again; the preoperative assessment was reviewed and updated.
5. Standard preoperative regimen including eyedrops, moxifloxacin hydrochloride 0.5% & ketorolac tromethamine 0.5%, 1 drop six hourly one day before surgery & eye drop tropic-amide 0.8% & phenylephrine hydrochloride 5%, 1 drop every fifteen minutes one hour before surgery. Peri-bulbar anaesthesia with 5-6ml lidocaine hydrochloride 2% plus adrenaline 1:200000 was given before surgery and pupillary dilation of 7-8mm was achieved.
6. All cataract surgeries were performed under peri-bulbar anaesthesia by a single experienced surgeon. Subjects underwent standard coaxial 2.75mm clear cornea phacoemulsification technique using in the bag one-piece acrylic posterior chamber intraocular lens implantation.
7. All participants were followed up and corneal endothelium parameters were measured and recorded on postoperative days 1, 7, 30, and 90 months.

2.9. Statistical analysis plan

The primary outcome was the difference in the endothelial morphology during the postoperative follow-up period among the diabetic and non-diabetic participants. We aimed to assess whether data supplied evidence for any statistical and clinically significant difference in the endothelial morphological parameters during various postoperative follow-up visits among the two comparison groups. All the data were collected in a paper-based data collection form. Thereafter, the data were coded and entered in Microsoft Excel. The coded data were imported into Stata 17.1 version for analysis. A comparison of continuous variables with baseline values was analysed using a student's t-test in each group. Categorical variables were analysed using chi-square (χ^2) tests. A P-value <0.05 was considered statistically significant. All tests are two-sided; the nominal level of type I error will be 5% and the confidence level for all confidence intervals will be 95%.

2.10. Null hypothesis

There is no significant difference in the various corneal endothelial morphological parameters after phacoemulsification for cataracts among diabetic and non-diabetic participants.

2.11. Alternative hypothesis

There is a significant difference in the various corneal endothelial morphological parameters after phacoemulsification for cataracts among diabetic and non-diabetic participants.

3. Results

To recruit the participants for the present study, the principal investigator approached a total of 238 participants; 22 patients were excluded using the selection criteria and 16 patients refused to participate (unable to commit to follow-up). A total of 200 participants were enrolled in the present study: 100 participants had diabetes (Group I) and 100 participants were non-diabetic (Group II). All participants completed the follow-up and there were no losses to follow up.

The mean age of the diabetic and non-diabetic participants was 65.5 and 64.6 years, respectively (p=0.448). The age of the participants in the study ranged from a minimum of 50 years to a maximum of 80 years. Overall, there were 102 (51%) female and 98 (49%) male participants. Overall, only 36 (18%) participants: 13 diabetic and 23 non-diabetics had no comorbidities. Further, the most common comorbidity was chronic kidney disease (20%), followed by Hypertension and cardiovascular diseases, 17.5% each. The mean BMI of the diabetic and non-diabetic patients was 27.8 and 25.4 kg/m^2 respectively (p=0.031). Overall, 51% of participants had cataracts in the left eye and the remaining 49% of participants had cataracts in the right eye. Most of the participants had Grade-2 cataracts (52.5%) and Grade-3 (47.5%).

Table 1: Socio-demographic characteristics of the participants (n=200)

Age Group	Group		P-value
	Diabetic (n=100)	**Non- Diabetic (n=100)**	
50-60	35 (35.0)	33 (33.0)	
61-70	31 (31.0)	37 (37.0)	-
71- 80	34 (34.0)	30 (30.0)	
Mean	65.5	64.6	0.448
Gender			
Female	46 (46.0)	56 (56.0)	0.1572
Male	54 (54.0)	44 (44.0)	
Obese	36 (36.0)	28 (28.0)	-
BMI	27.8	25.4	0.031
Right Eye	48 (48.0)	50 (50.0)	0.7773
Left Eye	52 (52.0)	50 (50.0)	
Grade of Cataract			
II	51 (51.0)	54(54.0)	0.671
III	49 (49.0)	46(46.0)	

Table 2: Diabetes-related parameters

Parameter	Diabetic (n=100)
Random Blood Sugar	179.2 (27.7)
Hb1AC	6.9 (0.72)
Duration of Diabetes	
< 5 years	19
5-10 Years	66
>10 Years	15
Mean	7.1 (±2.9)

Table 2 shows the parameters related to diabetes among the participants. The mean duration of diabetes among participants was 7.1 years, ranging from a minimum of 3.5 years to a maximum of 12 years. Lastly, most of the participants had diabetes for 5 to 10 years.

Table 3 shows during the preoperative period, the endothelial density among diabetic and non-diabetic patients was 2845 and 2915 (p = 0.873; not significant). Following cataract surgery, the ECD decreased among patients of both groups. However, the decline in the ECD was significantly higher (p<0.05) among diabetic patients. At each postoperative follow-up period, the ECD was significantly lower among diabetic patients in comparison to non-diabetic patients (p<0.05). Figure 1 illustrates the percentage change in endothelial cell density among the participants. The percentage change in ECD on day 1-, 7-, 30-, and 90 days of cataract surgery among the diabetic patients was 8.8%, 11.7%, 12.9% and 18.7%, respectively. The percentage change in ECD on day 1-, 7-, 30-, and 90

Table 3: Endothelial cell density

Duration	DM	Non- DM	P-value
Preoperative	2845 (±202.3)	2915 (±191.3)	0.873
Day 1	2594 (±234.8)	2792 (±229.3)	0.043
Day 7	2511 (±229.4)	2705 (±218.6)	0.033
Day 30	2478 (±231.9)	2647 (±207.6)	0.002
Day 90	2314 (±219.6)	2586 (±196.6)	0.08

days of cataract surgery among the non-diabetic patients was 5.3%, 7.2%, 9.2% and 11.3%, respectively.

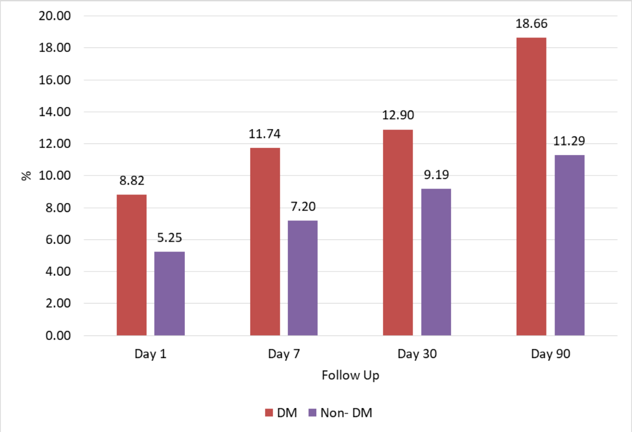

Figure 1: % change in ECD

Table 4 shows during the preoperative period, the ECH was slightly higher among non-diabetic patients (69.70 and 63.7; p-value = 0.089; not significant). Immediately following cataract surgery, the ECH declined among patients of both groups and later increased after seven days after cataract surgery. However, the decline in the ECH was significantly higher (p<0.05) among diabetic patients. At each postoperative follow-up period, the ECH was significantly lower among diabetic patients in comparison to non-diabetic patients (p<0.05).

Table 5 shows during the preoperative period, the CCT among diabetic and non-diabetic patients was almost equal (513 and 508; p-value = 0.083; not significant). Following cataract surgery, the CCT increased among patients of both groups. However, the increase in the CCT was significantly higher (p<0.05) among diabetic patients. At each postoperativefollow-up period, the central cornea was significantly thicker among diabetic patients in comparison to non-diabetic patients (p<0.05).

4. Discussion

The results of the present study suggest that diabetes damages corneal endothelium cell health. It was revealed that diabetes mellitus could reduce endothelial cell density. In addition, our results also revealed that diabetes mellitus could reduce the percentage of hexagonal cells, thus increasing the coefficient of variation of cell area. With corneal endothelium cells decreasing, the remaining corneal endothelium cells would be filled by the expansion and migration of neighbouring cells. Our study did not include patients with very hard nuclei (grade IV and above) because of the high energy required with such hard cataracts.

The existing literature suggests that if ECD falls below a threshold of 400–500 cells/mm2, at which point the cornea cannot maintain its normal physiological function. An ECD of 1000 cells/mm2 is typically regarded as the minimal preoperative value to avoid corneal decompensation after surgery.[24] Following cataract surgery, the ECD decreased among patients of both groups. However, the decline in the ECD was significantly higher (p<0.05) among diabetic patients. At each postoperative follow-up period, the ECD was significantly lower among diabetic patients in comparison to non-diabetic patients (p<0.05). Similar to our findings, a recent study by Dhasmana et al. revealed a substantial decline in ECD in the DM group following cataract surgery compared to the control.[25] Tang YE et al., conducted a meta-analysis of observational studies and concluded that diabetic patients are more prone to corneal endothelial damage after phacoemulsification, with DM patients having substantially higher endothelial cell loss than non-DM patients from the first day to three months after surgery (P =0.01).[26] Sahu PK et al., reported that patients with diabetes showed a significantly higher loss in endothelial cell count as compared to non-diabetic controls.[12] Furthermore, the patients with diabetes showed a slower recovery trend in the endothelial healing response as evidenced by a lower change in the coefficient of variation. Joo JH and Kim TG et al., reported that the mean change in ECD 1year after surgery was 13.28% in the DM group and 11.40% in the control group. In the fellow non-operated eyes, the mean change was 4.47% and 3.63% in the DM and control groups, respectively.[27]

The change in endothelial cell density is frequently used to assess the condition of corneas following phacoemulsification, although ECD is unable to capture the dynamics of the healing process. The dynamic of the corneal recovery process has a stronger association with the change in EC morphology. The endothelium's cells cannot regenerate as the corneal epithelium can. Instead, the surviving endothelial cells fill the area by enlarging and stretching to cover the posterior corneal surface. The ideal sequence of events would be a rise in cell size

Table 4: Endothelial cell hexagonality

Duration	DM	Non- DM	P-value
Preoperative	63.7 (±4.9)	69.7 (±4.1)	0.089
Day 1	52.1 (±5.3)	60.9 (±5.1)	0.021
Day 7	54.7 (±5.7)	61.7 (±4.8)	0.008
Day 30	56.8 (±4.8)	63.4 (±4.7)	0.014
Day 90	58.5 (±4.4)	67.6 (±4.6)	0.029

Table 5: Central corneal thickness

Duration	DM	Non- DM	P-value
Preoperative	513.27 (±17.5)	508.43 (±16.8)	0.083
Day 1	520.08 (±21.4)	514.59 (±21.3)	0.037
Day 7	524.93 (±22.7)	519.51 (±19.5)	0.029
Day 30	522.63 (±19.8)	517.11 (±18.3)	0.013
Day 90	524.54 (±18.3)	520.13 (±18.1)	0.018

accompanied by an expansion of CV and a drop in the percentage of Hexagonal Cells%. In the present study, immediately following cataract surgery, the ECH declined among patients of both groups and later increased seven days after cataract surgery. However, the decline in the ECH was significantly higher ($p<0.05$) among diabetic patients. At each postoperative follow-up period, the ECH was significantly lower among diabetic patients in comparison to non-diabetic patients ($p<0.05$). In the present study, postoperatively, there was consistently as well as significant difference in HC% loss between the DM and non-DM groups.

Tang Y et al., conducted a meta-analysis of 11 observational studies reporting the outcome of hexagonal cells. The reported that diabetic patients have a significantly smaller HC% at preoperative and all postoperative time points (all: $P < 0.001$) and a significantly larger HC% loss (difference of preoperative and postoperative) at all postoperative times (all: $P < 0.001$) compared to the non-DM group.[26]Similar findings were reported by Li et al.[28] and Kang K et al.,[29] but their study had a follow-up of only 1 month. In addition, Hugod et al.[30] showed a significant decline in the percentage of hexagonal cells among patients with diabetes only. Sahu PK et al., reported that patients with diabetes showed a slower recovery trend in the endothelial healing response as evidenced by the lower change in the ECH.[12] Similar to our findings, the authors also reported that the long disease-duration DM group (≥10years) had a significantly greater ECD loss than the control and short disease-duration DM groups (<10 years). Joo JH et al., reported no significant difference in the hexagonality between the diabetic and the non-diabetic patients.[27] Budiman B et al., reported no statistically significant differences in the CV and ECH between the diabetic and non-diabetic group at four-week follow-up after phacoemulsification.[31] Elminshawy AI et al., reported significant differences between preoperative and postoperative periods in both groups regarding coefficient of variation, and hexagonal cells, except those changes became insignificant with respect to CCT at 3 months in nondiabetic and at 1 week and 3 months in diabetic and in CD at 1 month in healthy patients.[32]

The healthy corneal endothelium is essential for preserving the cornea's moisture content and transparency as well as its integrity to prevent stromal oedema. An increase in the CCT results from failure of either the anatomical barrier or the pump function of the corneal endothelial cells leading to corneal edema. The intensity of the physical damage and the pathological insults caused by DM have an impact on CCT. In the present study, the CCT among diabetic and non-diabetic patients was almost equal (513 and 508; p-value = 0.083; not significant) during the preoperative period. Following cataract surgery, the CCT increased among patients of both groups. However, the increase in the CCT was significantly higher ($p<0.05$) among diabetic patients. At each postoperative follow-up period, the central cornea was significantly thicker among diabetic patients in comparison to non-diabetic patients ($p<0.05$).

According to the meta-analysis by Tang Y et al., the CCT of the DM group was significantly higher than that of the non-DM group at all postoperative time points.[26] Moreover, the percentage increase of CCT (dCCT%), showed a significant difference between the DM group and the non-DM group at 1 day postoperatively, 1 week postoperatively and 3 months postoperatively. The highest significant dCCT% was found in diabetic patients at 1 month postoperatively compared to the non-diabetic ones. Wang J t al. reported that the rise in CCT following phacoemulsification peaked at 1 day and 1 week postoperatively and subsequently steadily reduced for at least 3 months.[17] Nevertheless, compared to healthy controls, diabetics' postoperative corneal oedema healing was delayed. According to Chen ZF et al., although both diabetic and non-diabetic patients had greater corneal thicknesses one week after surgery, however, there were significant differences in corneal thickness among diabetic and non-diabetic patients.[33] Elkady MS et al.[21] reported that in terms of CCT, among diabetic patients the difference between preoperative CCT and 6 months postoperative was 3.3 (±0.95) micron.[21] In comparison among non-diabetic patients, the difference between preoperative CCT and 6 months postoperative was 2.73 (±1.64) microns. The authors concluded that corneal endothelium in diabetic subjects is more vulnerable to surgical trauma and has a lower capability in the process of repair.

5. Conclusion

In comparison to non-diabetic patients, there was a considerable loss of endothelial cell function among diabetic individuals following cataract surgery. To summarize, our study has found that patients with diabetes show a significantly higher loss of corneal endothelial density after cataract surgery as compared to nondiabetic controls. Furthermore, a slower and poor healing response was seen in patients with diabetes as evidenced by higher coefficient of variation and lower endothelial cell hexagonality. These changes were seen even in the presence of good glycaemic control, thus other factors might be contributing to the increased vulnerability of endothelial cells in patients with diabetes. This shows that diabetic patients' eyes are under metabolic stress and have less reserve capacity than non-diabetic eyes' corneal endothelium. Therefore, in patients with diabetes, surgery should be scheduled with the proper safety measures. In light of these findings, specular microscopy should be performed on every type II diabetic patient prior to scheduling cataract surgery.

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7. Conflict of Interest

None.

References

1. Tuft SJ, Coster DJ. The corneal endothelium. *Eye*. 1990;4(3):389–91.
2. Delmonte DW, Kim T. Anatomy and physiology of the cornea. *J Cataract Refract Surg*. 2011;37(3):588–98.
3. Zavala J, Jaime GRL, Barrientos CAR, Valdez-Garcia J. Corneal endothelium: Developmental strategies for regeneration. *Eye (Lond)*. 2013;27(5):579–88.
4. Eghrari AO, Riazuddin SA, Gottsch JD. Overview of the cornea: structure, function, and development. *Prog Mol Biol Transl Sci*. 2015;134:7–23.
5. Mishima S. Clinical investigations on the corneal endothelium-XXXVIII Edward Jackson Memorial Lecture. *Am J Ophthalmol*. 1982;93(1):1–29.
6. Gupta PK, Berdahl JP, Chan CC, Rocha KM, Yeu E, Ayres B, et al. The corneal endothelium: clinical review of endothelial cell health and function. *J Cataract Refract Surg*. 2021;47(9):1218–26.
7. Bourne WM. Biology of the corneal endothelium in health and disease. *Eye (Lond)*. 2003;17(8):912–8.
8. Taurone S, Miglietta S, Spoletini M, Feher J, Artico M, Papa V, et al. Age related changes seen in human cornea in formalin fixed sections and on biomicroscopy in living subjects: A comparison. *Clin Anat*. 2020;33(2):245–56.
9. Muir KW, Duncan L, Enyedi LB, Stinnett SS, Freedman SF. Central corneal thickness in children: stability over time. *Am J Ophthalmol*. 2006;141(5):955–7.
10. Foster A, Frcophth F. Vision 2020: the cataract challenge. *Community Eye Health*. 2000;13(34):17–9.
11. Burton MJ, Ramke J, Marques AP, Bourne RRA, Congdon N, Jones I, et al. The Lancet Global Health Commission on Global Eye Health: vision beyond 2020. *Lancet Glob Health*. 2021;9(4):489–551.
12. Sahu PK, Das GK, Agrawal S, Kumar S. Comparative evaluation of corneal endothelium in patients with diabetes undergoing phacoemulsification. *Middle East Afr J Ophthalmol*. 2017;24(2):74–80.
13. Shoham A, Hadziahmetovic M, Dunaief JL, Mydlarski MB, Schipper HM. Oxidative stress in diseases of the human cornea. *Free Radic Biol Med*. 2008;45(8):1047–55.
14. Choi JY, Han YK. Long-term (≥10 years) results of corneal endothelial cell loss after cataract surgery. *Can J Ophthalmol*. 2019;54(4):438–44.
15. Ganesan N, Srinivasan R, Babu KR, Vallinayagam M. Risk factors for endothelial cell damage in diabetics after phacoemulsification. *Oman J Ophthalmol*. 2019;12(2):94–8.
16. Beato JN, Esteves-Leandro J, Reis D, Falcão M, Rosas V, Carneiro A, et al. Corneal structure and endothelial morphological changes after uneventful phacoemulsification in type 2 diabetic and nondiabetic patients. *Arq Bras Oftalmol*. 2021;84(5):454–61.
17. Wang B, Li JX, Wang YL, Wu BG, Huo JX. Clinical effect analysis of phacoemulsification on cataract patients with diabetes mellitus. *Int Eye Science*. 2013;13:1163–6.
18. Ganesan N, Srinivasan R, Babu KR, Vallinayagam M. Risk factors for endothelial cell damage in diabetics after phacoemulsification. *Oman J Ophthalmol*. 2019;12(2):94–8.
19. Beato JN, Esteves-Leandro J, Reis D, Falcão M, Rosas V, Carneiro A, et al. Corneal structure and endothelial morphological changes after uneventful phacoemulsification in. *Arq Bras Oftalmol*. 2021;84(5):454–61.
20. Sahu PK, Das GK, Agrawal S, Kumar S. Comparative Evaluation of Corneal Endothelium in Patients with Diabetes Undergoing Phacoemulsification. *Middle East Afr J Ophthalmol*. 2017;24(2):74–80.
21. Elkady MS, Saleh MM, Aboalhamd AS. Corneal Endotheleal Cells Changes after Phacoemulsification in Type II Diabetes Mellitus. *Egypt J Hosp Med*. 2017;69(3):2008–15.
22. Chen ML. Effect of phacoemulsification on the corneal endothelium of diabetic patients with cataract. *Int Eye Sci*. 2018;18:1786–91.
23. Zhao G. Changes of corneal endothelium in diabetes patients after cataract phacoemulsification surgery by confocal microscopy. *Int Eye Sci*. 2013;13:876–9.
24. Inoue K, Kato S, Inoue Y, Amano S, Oshika T. The corneal endothelium and thickness in type II diabetes mellitus. *Jpn J Ophthalmol*. 2002;46(1):65–9.
25. Dhasmana R, Singh IP, Nagpal RC. Corneal Changes in Diabetic Patients after Manual Small Incision Cataract Surgery. *J Clin Diagn Res*. 2014;8(4):VC03.
26. Tang Y, Chen X, Zhang X, Tang Q, Liu S, Yao K. Clinical evaluation of corneal changes aftr phacoemulsification in diabetic and non-diabetic cataract patients, a systematic review and meta-analysis. *Sci Rep*. 2017;7(1):14128.
27. Joo JH, Kim TG. Comparison of corneal endothelial cell changes after phacoemulsification between type 2 diabetic and nondiabetic patients. *Medicine (Baltimore)*. 2021;100(35):e27141.

28. Li MH, Fu XL, Yang WF. Effect and risk factors for corneal endothelial cells after phacoemulsification in diabetic cataract patients. *Int Eye Sci*. 2016;16:1048–51.
29. Kang KH, Song MY, Kim KY, Hwang KY, Kwon YA, Koh K. Corneal endothelial cell changes after femtosecond laser-assisted cataract surgery in diabetic and nondiabetic patients. *Eye Contact Lens*. 2021;47(12):664–9.
30. Hugod M, Storr-Paulsen A, Norregaard JC, Nicolini J, Larsen AB, Thulesen J. Corneal endothelial cell changes associated with cataract surgery in patients with type 2 diabetes mellitus. *Cornea*. 2011;30(7):749–53.
31. Budiman B. Comparison of endothelial cell density, morphological changes and central corneal thickness after phacoemulsification between diabetic and non-diabetic patients. *Open Ophthalmol J*. 2020;14(1):15–20.
32. Elminshawy AI, Saiedb HH, Mohamad AM. Comparison of corneal changes after phacoemulsification in diabetic and nondiabetic eyes. *Al-Azhar Assiut Med J*. 2022;20(1):134–41.
33. Chen Z, Song F, Sun L, Zhao C, Gao N, Liu P, et al. Corneal integrity and thickness of central fovea after phacoemulsification surgery in diabetic and nondiabetic cataract patients. *Arch Med Sci*. 2018;14(4):818–25.

Author's biography

Prerna Upadhyaya, Cornea Head/Medical Director

Aishwarya Singh Raghuvanshi, DNB Resident

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