Mini Review

iMedPub Journals
http://www.imedpub.com

Insights in Ophthalmology

2017
Vol. 1 No. 2: 8

Corneal Endothelium and Central Corneal Thickness Changes in Type 2 Diabetes Mellitus

Amira El-Agamy

Mansoura Ophthalmic Center, Faculty of Medicine, Mansoura University, Egypt

Received: April 10, 2017; **Accepted:** April 24, 2017; **Published:** April 29, 2017

Corresponding author:
Amira El-Agamy, M.D.

aelagamy@KSU.EDU.SA

Department of Optometry and Vision Sciences, College of Applied Medical Sciences, King Saud University, Saudi Arabia and Mansoura Ophthalmic Center, Faculty of Medicine, Mansoura University, Egypt.

Tel: + 00966118051475

Citation: El-Agamy A. Corneal Endothelium and Central Corneal Thickness Changes in Type 2 Diabetes Mellitus. Ins Ophthal. 2017, 1:2.

Review of Literature

It is predicted that the number of diabetic patients will reach almost 552 million by the year 2030 [1]. Type 2 diabetes mellitus (DM) represents 90% of cases and is characterized by overweight and insulin resistance [2]. It is found that nearly 20% of patients identified by type 2 DM actually have type 1.5, or latent autoimmune DM who is a form of type 1 DM that affects adults with a slower course of onset compared to type 1 DM [2,3]. This special group of diabetic patients is not overweight and does not show insulin resistance [2].

All corneal layers are involved in DM in different ways such as corneal endothelial damage, recurrent corneal erosions, diminished corneal sensitivity, ulcers, delayed wound healing, and superficial keratitis [4-7]. Folds in Descemet's membrane were detected earlier in diabetic patients compared to non-diabetic elderly people [8-11].

Choo et al. [12] study documented morphological anomalies of corneal endothelium in type 2 diabetics such as a reduced endothelial cell density (ECD), polymorphism (decrease in the percentage of hexagonal cells), in addition to polymegathism (increased coefficient of variation (CV) of cell area (standard CV values are between 0.22 and 0.31 and beyond 0.4 is unusual).

Tripathy et al. [13] Choo et al. [12] and Browning [8] studies demonstrated that increased glucose levels induce augmented activity of the aldose reductase enzyme leading to sorbitol accumulation in the corneal epithelial and endothelial cells, which behaves as an osmotic agent producing swelling of corneal endothelium in diabetic cornea. Moreover, Na+ – K+ ATPase activity of the corneal endothelium in diabetics is decreased causing alterations of the corneal morphological and permeability features [14] In addition, diminished ATP manufacture caused by slowing down of the Krebs cycle disturbs endothelial pump function in diabetic cornea [12].

El-Agamy and Alsubaie [15], Sudhir et al. [16], Choo et al. [12], Inoue et al. [17] and Saini and Mittal [18] studies documented a significant reduction in ECD of diabetic corneas compared to controls. Conversely, Siribunkum et al. [19] study showed a significant increased corneal endothelial cell density in diabetic cornea. On the other hand, Storr-Paulsen et al. [20] confirmed that controlled type II DM has no influence on ECD.

El-Agamy and Alsubaie [15], Shenoy et al. [21] and Lee et al. [22] studies showed a significant polymegathism in diabetic cornea. In opposition, Chen et al. [23] and Sudhir et al. [16] studies did not detect any significant polymegathism in diabetic cornea compared to non-diabetics.

Choo et al. [12] and Lee et al. [22] studies found a significant polymorphism in diabetic corneas compared to controls. Alternatively, El-Agamy and Alsubaie [15], Storr-Paulsen et al. [20], Sudhir et al. [16] and Inoue et al. [17] studies demonstrated no significant difference between the two groups.

Storr-Paulsen et al. [20], Su et al. [24] and Lee et al. [22] studies showed a significant increase in central corneal thickness (CCT) of diabetic cornea compared to controls. While El-Agamy and Alsubaie [15], Sudhir et al. [16] and Choo et al [12] studies did not show any significant difference between the two groups. Su et al. [24] and Busted et al. [25] studies proposed that increased CCT may be one of the earliest demonstrable alterations in diabetic eyes.

Regarding evaluation of the mean values of CCT, ECD, CV and cells hexagonality in patients with DM duration ≤ 10 years and those with DM duration >10 years: El-Agamy and Alsubaie [15] found no significant difference between the two groups. Also, Altay et al. [26] did not report any significant difference in CCT between the two groups. On the other hand, Lee et al. [22] found significantly higher CCT and CV in patients with DM duration >10 years than those with DM duration ≤ 10 years, without any significant difference regarding ECD and hexagonality between the two groups.

As regards assessment of the mean values of CCT, ECD, CV and cells hexagonality in diabetic patients with HbA1c ≤ 7.5% and those with HbA1c>7.5%: El-Agamy and Alsubaie [15] reported a significant difference only in CV between the two groups. However, Storr-Paulsen et al. [20] demonstrated lower ECD in patients with elevated HbA1c without any significant difference in CCT between the two groups. On the other hand, Altay et al. [26] found a significant increase in CCT in uncontrolled diabetics.

Shenoy et al. [21] and Saini and Mittal [18] found significantly lower ECD in eyes with diabetic retinopathy (DR) compared to those without DR. Moreover, Busted et al. [25] documented association of increased HbA1c and blood glucose levels, and severe retinal complications with increased CCT. Conversely, El-Agamy and Alsubaie [15] demonstrated no significant difference.

Conclusion

Some studies documented significant corneal changes in type 2 diabetic patients compared to non-diabetics whereas other studies did not find any significant differences between the two groups. Future studies with a large sample size and for long follow up duration will elucidate the scope of corneal insult caused by type 2 DM.

Acknowledgement

This research project was supported by a grant from the “Research Center of the Female Scientific and Medical Colleges”, Deanship of Scientific Research, King Saud University.

References

1 Agarwal A, Soliman MK, Sepah YJ, Do DV, Nguyen QD (2014) Diabetic retinopathy: Variations in patient therapeutic outcomes and pharmacogenomics. Pharmgenomics Pers Med 7: 399-409.

2 Kathryn Skarbez, Yos Priestley, Marcia Hoepf, Steven B Koevary (2010) Comprehensive review of the effects of diabetes on ocular health. Expert Rev Ophthalmol 5: 557-577.

3 Stenström G, Gottsäter A, Bakhtadze E, Berger B, Sundkvist G (2005) Latent autoimmune diabetes in adults: Definition, prevalence, beta-cell function and treatment. National Center for Biotechnology Information. PMID 16306343.

4 Shih KC, Lam KS, Tong L (2017) A systematic review on the impact of diabetes mellitus on the ocular surface. Nutr Diabetes 7: e251.

5 Thomas N, Jeyaraman K, Asha HS, Velevan J (2012) A practical guide to diabetes mellitus. Jaypee Brothers Medical Pub.

6 Saini JS, Khandalavla B (1995) Corneal epithelial fragility in diabetes mellitus. Can J Ophthalmol 30: 142-146.

7 Herse PR (1988) A review of manifestations of diabetes mellitus in the anterior eye and cornea. Am J Optom Physiol Optics 65: 224-230.

8 Browning DJ (2010) Diabetic retinopathy: Evidence-based management. Springer, New York.

9 Mocan MC, Irkec M, Orhan M (2006) Evidence of Waite-Beetham lines in the corneas of diabetic patients as detected by *in vivo* confocal microscopy. Eye 20: 1488-1490.

10 Rubenstein MP (1987) Diabetes, the anterior segment and contact lens wear. Contact Lens J 15: 4-11.

11 Henkind P, Wise GN (1961) Decemet's wrinkles in diabetes. Am J Ophthalmol 52: 371-374.

12 Choo M, Prakash K, Samsudin A, Soong T, Ramli N, et al. (2010) Corneal changes in type II diabetes mellitus in Malaysia. Int J Ophthalmol 3: 234-236.

13 Tripathy BB, Chandalia HB, Das AK, Rao PV (2012) Textbook of diabetes mellitus. Jaypee Brothers Medical Publishers.

14 Herse PR (1990) Corneal hydration control in normal and alloxan-induced diabetic rabbits. Invest Ophthalmol Vis Sci 31: 2205-2213.

15 El-Agamy A, Alsubaie S (2017) Corneal endothelium and central corneal thickness changes in type 2 diabetes mellitus. Clin Ophthalmol 11: 481-486.

16 Sudhir R, Raman R, Sharma T (2012) Changes in the corneal endothelial cell density and morphology in patients with type 2 diabetes mellitus: A population-based study, Sankara Nethralaya Diabetic Retinopathy and Molecular Genetics Study (SN-DREAMS, Report 23). Cornea 31: 1119-1122.

17 Inoue K, Kato S, Inoue Y, Amano S, Oshika T (2002) The corneal endothelium and thickness in type II diabetes mellitus. Jpn J Ophthalmol 46: 65-69.

18 Saini JS, Mittal S (1996) In vivo assessment of corneal endothelial function in diabetes mellitus. Arch Ophthalmol 114: 649-653.

19 Siribunkum J, Kosrirukvongs P, Singalavanija A (2001) Corneal abnormalities in diabetes. J Med Assoc Thai 84: 1075-1083.

20 Storr-Paulsen A, Singh A, Jeppesen H, Norregaard J, Thulesen J (2013) Corneal endothelial morphology and central thickness in patients with type II diabetes mellitus. Acta Ophthalmologica 92: 158-160.

21 Shenoy R, Khandekar R, Bialasiewicz A, Al Muniri A (2009) Corneal endothelium in patients with diabetes mellitus: A historical cohort study. Eur J Ophthalmol 19: 369-375.

22 Lee JS, OumBS, Choi HY, Lee JE, Cho BM (2006) Differences in corneal thickness and corneal endothelium related to duration in diabetes. Eye 20: 315-318.

23 Chen Y, Huang S, Jonna G, Channa P (2014) Corneal endothelial cell changes in diabetes mellitus. Invest Ophthalmol Vis Sci 55: 2054.

24 Su DHW, Wong TY, Wong W (2008) Diabetes, hyperglycemia and central corneal thickness. Ophthalmology 115: 964-968.

25 Busted N, Olsen T, Schmitz O (1981) Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus. Br J Ophthalmol 65: 687-690.

26 Altay Y, Burcu A, Ornek F (2014) The change in central corneal thickness after successful control of hyperglycemia in diabetic patients. Int Eye Sci 14: 575-578.