ORIGINAL RESEARCH PAPER

Volume-8 | Issue-3 | March-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

A STUDY ON ASSOCIATION BETWEEN DIABETES MELLITUS AND CENTRAL CORNEAL THICKNESS

Ophthalmology

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ABSTRACT

Background: As the prevalence of type 2 diabetes mellitus rises, so do its attendant, micro vascular complications. Besides, diabetic retinopathy, patients with diabetes are prone to developing corneal endothelial damage, keratoepitheliopathy in the form of recurrent corneal erosions, persistent epithelial defects, and superficial keratitis.

Objectives: To find out the correlation between Diabetes mellitus and Central corneal thickness (CCT).

Material and Methods: It was a hospital based, Case-Control study. A total of 100 patients, 40-70yrs of age, participated in the study, of whom 50 were diabetic and 50 were non-diabetic. Consenting patients of age 40-70yrs was done, attending Ophthalmology OPD of medical college in Lucknow, who did not have any corneal pathology were divided into two groups – diabetic and non-diabetic. After thorough local and systemic examination, the central corneal thickness was measured using Ultrasound pachymetry.

Results: Mean CCT was thicker in diabetic group (575.53µm) when compared to non-diabetic group (560.40µm). The difference between the 2 groups was significant ($p<0.001$). Mean CCT in diabetic patients was 575.53±7.8µm. Mean duration of diabetes was 9.53±1.9 yrs. The correlation between CCT and duration of diabetes was not significant ($p=0.158$).

Conclusion: The CCT in diabetic patients was thicker compared to CCT in non–diabetics. Therefore, diabetes can increase the CCT but there is no significant correlation between duration of diabetes and CCT.

KEYWORDS

Central Corneal thickness, Pachymetry.

INTRODUCTION:

Corneal endothelium plays a major role in maintaining the optical transparency of the cornea. Extrinsic factors, such as genetics, race, and age,[1-6] or intrinsic factors, such as trauma, intraocular surgery, ultraviolet radiation, infection, etc,[3,7-10] are responsible for maintaining the structural and functional integrity of the corneal endothelium.

Diabetes mellitus is one of the most common non-communicable diseases globally. At the ocular level, main indicators of diabetes are retinopathy, cataract and glaucoma[11].Diabetic keratopathy is a frequent disease that entails several alterations, especially in the epithelium and endothelium, like punctate epithelial keratopathy, recurrent corneal erosions and persistent epithelial defects. Diabetic keratopathy can cause alterations in all layers of cornea especially the endothelium like decrease in endothelial cell density and hexagonality, as well as increased polymegathism, pleomorphism and central corneal thickness[12-13]. As the prevalence of type 2 diabetes mellitus rises, so do its attendant, microvascular complications.[14] Besides, diabetic retinopathy, patients with diabetes are prone to developing corneal endothelial damage, keratoepitheliopathy in the form of recurrent corneal erosions, persistent epithelial defects, and superficial keratitis.[15-17]

Central corneal thickness measurements are vitally important for the diagnosis, treatment and management of various ocular conditions. Corneal pachymetry measures corneal thickness, a sensitive indicator of endothelial physiology that correlates well with functional measurements.[18] Techniques for measuring central corneal thickness include optical pachymetry, ultrasound pachymetry, confocal microscopy, ultrasound biomicroscopy, optical ray path analysis or scanning slit corneal topography and optical coherence tomography[19].Ultrasound pachymetry is the current standard for corneal thickness measurement[20].

Although morphological and physiological changes in the corneal endothelium, in patients with diabetes, have been documented, most of the studies had small sample size, and no such changes were reported from the large population-based sample.[21-26]

AIMS AND OBJECTIVES:

The aim of this study was to measure the central corneal thickness in diabetic and non-diabetic individuals using ultrasound pachymetry and to evaluate the correlation between central corneal thickness and diabetes.

MATERIAL AND METHODS:

This study was designed as a case-control study conducted in Department of Ophthalmology of Era's Lucknow medical college, for duration of 1 year. Individuals aged 40-70 years attending Eye OPD were included. Exclusion criteria included patients with history of intraocular surgery, trauma, contact lens wear and receiving treatment for any topical or systemic diseases, patients with underlying ocular pathology and those who refused to give consent.

Sample size was 100 patients - 50 diabetics: 50 non-diabetics. Complete medical history, detailed local and systemic examination was done. CCT was measured using ultrasound pachymetry. Data was analysed using SPSS 17. Comparison between the different parameters was done using student t-test, Pearson correlation coefficient and Chi-square test. p-value<0.05 was considered significant.95% confidence limit was used.

RESULTS:

Mean CCT of 50 diabetic patients was 575.53 µm and that of non-diabetics was 560.40 µm. Mean CCT was thicker in diabetic groups when compared with the non-diabetics. The difference of the mean CCT between the 2 groups was found to be significant ($p<0.001$). Mean CCT was almost higher in males (570.67µm) than females (556.75µm) and the small difference was not significant ($p=0.176$). No correlation was found between CCT and gender. Mean CCT in diabetic patients were 575.53µm. Mean duration of diabetes was 9.53 yrs. We found no correlation between the CCT and duration of diabetes.The patients who had diabetes ≥ 10 years had higher CCT compared to those who had diabetes <10 years. This finding was not statistically significant ($p=0.095$).

Table 1: Mean central cornel thickness (CCT) amongst diabetics and non diabetics

Groups	Total no. of cases	Mean CCT(µm)	Std. Deviation	p-value	95% CI
Diabetic	50	575.53	42.33	<0.001	8.5-23.56
Non diabetic	50	560.40	21.56		

Table 2: CCT and duration of Diabetes Mellitus

Variable	No. of cases	Mean	Pearson's correlation	P-value
CCT	50	575.53	0.125	0.158
Duration of diabetes	50	9.53		

Table 3: CCT and Gender variation

Gender	Total no.	Mean CCT	Std deviation	p-value
Males	55	570.67	35.57	0.176
Females	45	556.75	30.68	

Table 4: Association of CCT with duration of diabetes

Duration of Diabetes in years	CCT		$\chi 2$	p-value
	<570µm	≥570µm		
<10	30	25	3.96	0.095
≥10	20	25		

DISCUSSION:

Diabetes reduces the activity of Na+K+ATPase of the corneal endothelium and this causes the morphological and functional changes of diabetic cornea[27]. Mean CCT of 50 diabetic patients were 575.53µm and that of non-diabetics were 560.40µm.The mean CCT was thicker in diabetic groups when compared with the non-diabetics. The difference between the 2 groups was statistically significant when analysed by t-test ($p<0.001$).Most studies and the present study showed that diabetic eyes had increased CCT when compared to non-diabetic subjects[28,29,30,31].M O Zengin et al postulated that endothelial pump function disturbance due to reduction of Na+K+ATPase activity results in an increase in stromal hydration[28,32]. N McNamara et al stated that CCT changes were due to hyperglycaemic effect on the cornea which directly inhibits the corneal endothelial pump. Other possible mechanisms that may account for the swelling differences between diabetics and non-diabetic subjects included reduced corneal lactate production and increased endothelial pump function during corneal hypoxia[33] Intracellular accumulation of sorbitol, which acts as an osmotic agent leads to swelling of the endothelial cells. The Kreb's cycle slows down with a consequent reduction in ATP production which is necessary for endothelial pump function. This eventually results in morphological and permeability changes in the cornea.

Limitations of study:

Further more elaborate study considering corneal changes in Diabetes Mellitus can be done involving multiple parameters and more than one centre.

CONCLUSION:

On the basis of this study it is concluded that an increase in central corneal thickness is present in early stages of diabetes. Diabetic patients exhibit a greater statistically significant average central corneal thickness than non-diabetics. Further, it is recommended to complement CCT findings with a parallel study of the corneal endothelium in these patients in order to assess whether there is a correlation between corneal thickness and conditions of the endothelium. Correlation with blood sugar level and HbA1 can also be measured along with the above mentioned parameters.

ACKNOWLEDGEMENT:

We are also thankful to junior doctors and staff of Ophthalmology department of Era's Lucknow Medical College and Hospital. Special thanks to everyone who participated in the study.

REFERENCES:

1. Mäkitie J, Vannas A, Koskenvuo M. Corneal endothelial cells in mono and di-zygotic twins. Invest Ophthalmol Vis Sci. 1983;24:1029–1032.
2. Matsuda M, Yee RW, Edelhauser HF. Comparison of the corneal endothelium in an American and a Japanese population. Arch Ophthalmol. 1985;103:68–70.
3. Bourne WM, Kaufman HE. Specular microscopy of human corneal endothelium in vivo. Am J Ophthalmol. 1976;81:319–323.
4. Laing RA, Sanstrom MM, Berrospi AR, et al. Changes in the corneal endothelium as a function of age. Exp Eye Res. 1976;22:587–594.
5. Rao SK, Ranjan Sen P, Fogla R, et al. Corneal endothelial cell density and morphology in normal Indian eyes. Cornea. 2000;19:820–823.
6. Yee RW, Matsuda M, Kern TS, et al. Corneal endothelial changes in diabetic dogs. Curr Eye Res. 1985;4:759–766.
7. Bourne WM, Nelson LR, Hodge DO. Continued endothelial cell loss ten years after lens implantation. Ophthalmology. 1994;101:1014–1022; discussion 1022–1023.
8. Slingsby JG, Forstot SL. Effect of blunt trauma on the corneal endothelium. Arch Ophthalmol. 1981;99:1041–1043.
9. Karai I, Matsumura S, Takise S, et al. Morphological change in the corneal endothelium due to ultraviolet radiation in welders. Br J Ophthalmol.1984;68:544–548.
10. Schultz RO, Glasser DB, Matsuda M, et al. Response of the corneal endothelium to cataract surgery. Arch Ophthalmol. 1986;104:1164–1169.
11. Fernandez-Vigo Lopez J. Diabetes Ocular LXVIII Ponencia official de la Sociedad Espanola de oftalmologia, Barcelona, EDIKA–MED, 1992
12. K Inoue,S Kato,Y Inoue,SAmano,TOshika,The corneal endothelium and thickness in type 2 diabetes mellitus,Jpn JOphthalmol46(1), 2002,65-9.
13. J Siribunkum ,PKosrirukvongs,ASingalavanija, Corneal abnormalities in diabetes, J Med Assoc Thai84(8),2001,1075-83.
14. Wild S, Roglic G, Green A, et al. Global prevalence of diabetes: estimates for the year 2000 and projection 2030. Diabetes Care. 2004; 27:1047–1053.
15. Schultz RO, Matsuda M, Yee RW, et al. Corneal endothelial changes in type I and type II diabetes mellitus. Am J Ophthalmol. 1984;98:401–410.
16. Schultz RO, Van Horn DL, Peters MA, et al. Diabetic keratopathy. Trans Am Ophthalmol Soc. 1981;79:180–199.
17. Herse PR. A review of manifestations of diabetes mellitus in the anterior eye and cornea. Am J Optom Physiol Opt. 1988;65:224–230.
18. A Farjo, M McDermontt, HK Soong, Corneal anatomy, physiology and wound healing. In: M Yanoff, JS Duker, editors.Ophthalmology(St.Louis:Mosby,2009)203-8.
19. V Yaylali, SC Kaufman, HW Thompson, Corneal thickness measurement with orbscan topography system and ultrasonic pachymetry, J Cataract Refract Surg23(9),1997,1345-50.
20. JJ Salz, SP Azen, J Berstein, P Caroline, RA Villasenor, DJ Schanzlin et al ,Evaluation and comparison of sources of variability in the measurement of corneal thickness with ultrasonic pachymeter, OphthalSurg14,1983,750-4.
21. Busted N, Olsen T, Schmitz O. Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus. Br J Ophthalmol. 1981;65:687–690.
22. Inoue K, Kato S, Inoue Y, et al. The corneal endothelium and thickness in type II diabetes mellitus. Jpn J Ophthalmol. 2002;46:65–69.
23. Itoi M, Nakamura T, Mizobe K, et al. Specular microscopic studies of the corneal endothelia of Japanese diabetics. Cornea. 1989;8:2–6.
24. Larsson LI, Bourne WM, Pach JM, et al. Structure and function of the corneal endothelium in diabetes mellitus type I and type II. Arch Ophthalmol. 1996;114:9–14.
25. Roszkowska AM, Tringali CG, Colosi P, et al. Corneal endothelium evaluation in type I and type II diabetes mellitus. Ophthalmologica. 1999;213:258–261.
26. Shenoy R, Khandekar R, Bialasiewicz A, et al. Corneal endothelium in patients with diabetes mellitus: a historical cohort study. Eur J Ophthalmol. 2009;19:369–375.
27. PR Herse, Corneal hydration control in normal and alloxan-induced diabetic rabbits, Invest Ophthalmol Vis Sci, 31(11),1990,2205-13.
28. N Busted ,T Olsen ,O Schmitz, Clinical observations on the corneal thickness and the corneal endothelium in diabetes mellitus,Br J Ophthalmol 65(10),1981,687-90.
29. JS Lee,BSOum, HY Choi, JE Lee, BM Cho, Difference in corneal thickness and corneal endothelium related to duration in diabetes, Eye 20(3),2006,315-8.
30. AM Roszkowska,CGTringali,PColosi,CASqueri,G Ferreri,Corneal endothelium evaluation in type1 and type2 diabetes mellitus,Ophthalmologica213(4),1999,258-61.
31. LI Larsson, WM Bourne, JM Pach, RF Brubaker, Structure and function of the corneal endothelium in diabetes mellitus type 1 and type 2, Arch Ophthalmol 114(1),1996,9-14.
32. ME Rosenberg ,TMTTervo,IJ Immonen,LJ Muller,CGronhagen-Riska,MHVesaluoma, Corneal structure andsensitivity in type 1 diabetes mellitus,Invest Ophthalmol Vis Sci41(10),2000,2915-21.
33. NA McNamara, RJ Brand, KA Poise and WM Bourne, Corneal function during normal and high serum glucose levels in Diabetes.IOVS 39(1),1998,3-17.