

Study on correlation between Diabetes mellitus and Central corneal thickness

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Abstract:

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

Design: Hospital based, Case-Control study.

Sampling And Sample Size: A total of 100 patients, 30-70yrs of age, participated in the study, of whom 50 were diabetic and 50 were non-diabetic.

Methods: Consenting patients of age 30-70yrs, attending RIMS OPD, 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 (574.359 μ m) when compared to non-diabetic group (557.76 μ m). The difference between the 2 groups was significant (p=0.000). Mean CCT in diabetic patients was 574.349 \pm 7.8 μ m. Mean duration of diabetes was 8.478 \pm 1.9 yrs. The correlation between CCT and duration of diabetes was not significant (p=0.238).

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

Keywords: Central Corneal thickness, Pachymetry.

I. Introduction

Cornea is the most important refractive element in the human ocular system, providing approximately two-third(40-45D) dioptres of the eye's power [1].In the frontal view the cornea appears elliptical, being 11.7mm wide horizontally and 10.6mm vertically. The posterior surface appears circular and is about 11.7mm in diameter[2].Corneal thickness is a sensitive indicator of health of cornea and serves as an index for corneal hydration and metabolism. It is also an important indicator of patency of corneal endothelium pump [3].Normal cornea has a central thickness of about 0.52mm and becomes thicker in paracentral zone and peripheral zone [4].

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[5].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 [6,7].

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[8].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[9].Ultrasound pachymetry is the current standard for corneal thickness measurement [10].The ultrasound pachymeter is designed for measuring the axial length of the eye and the thickness of the cornea. Ultrasound energy is emitted from the probe tip acting as both the transmitter and receiver. Some of the energy is reflected back towards the probe in the form of an echo. Measurement data can be calculated based on both the time it takes the echo to travel back to the probe from the eye and the preset converted velocity.

II. Aims And Objects

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.

III. Materials And Methods

This study was designed as a case-control study conducted in Department of Ophthalmology, RIMS for duration of 2years. Individuals aged 30-70years attending Eye OPD. 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 16. 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.

IV. Results

Mean age was 45.85 years. Median age was 45.50 yrs. 40% were in the age group of 41-50 years. 52% were males and 48% were females. Of the 50 diabetic cases, majority had diabetes of 1-5 years duration.

Mean CCT of 50 diabetic patients was 574.359 μ m and that of non-diabetics was 557.769 μ m. 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=0.000). Mean CCT was almost similar in males (568.489 μ m) and females (563.436 μ m) and the small difference was not significant (p=0.27). No correlation was found between CCT and gender (p=0.078). Mean CCT in diabetic patients were 574.359 μ m. Mean duration of diabetes was 8.478 yrs. We found no correlation between the CCT and duration of diabetes (p=0.238). The patients who had diabetes \geq 10 years had higher CCT compared to those who had diabetes <10 years. This finding was not statistically significant (p=0.139).

V. Figures And Tables

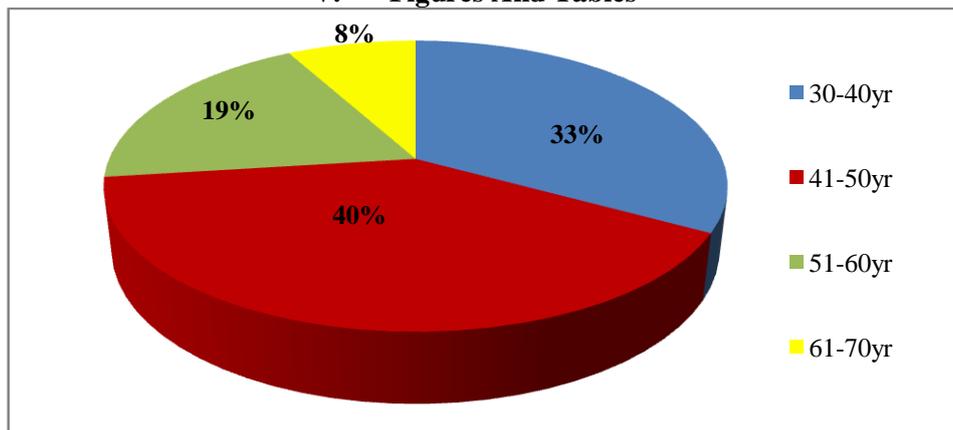


Fig 1: Distribution of patients by age.

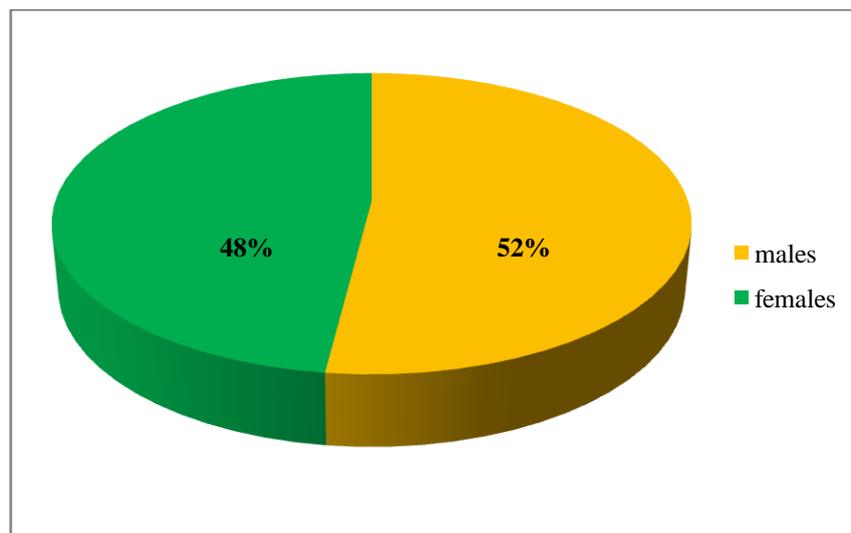


Fig.2: Gender distribution

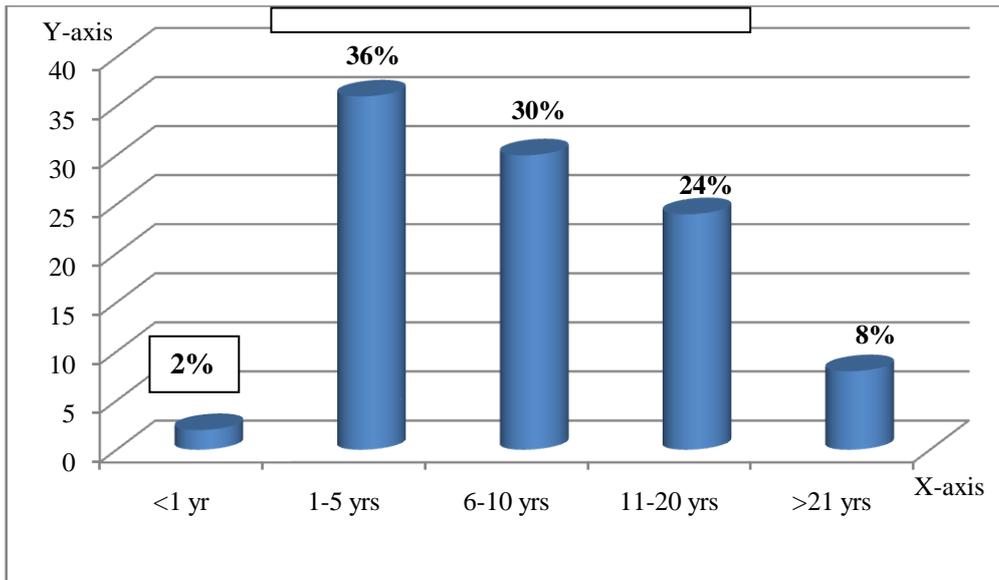


Fig 3: Diagram showing distribution of diabetic cases and duration of diabetes.

TABLE 1: Mean Central Corneal thickness

Groups	Total no of cases	Mean CCT(μm)	Std deviation	P- value	95% CI
DIABETIC	50	574.359	39.157	0.000	7.8-25.36
NON - DIABETIC	50	557.769	20.959		

TABLE 2: CCT and Duration of diabetes

Variable	Total no. of cases	Mean	Pearson correlation (r^2)	P- value
CCT	50	574.359(μm)	0.119	0.238
DURATION OF DIABETES	50	8.478(YRS)		

TABLE 3: CCT and Gender

Gender	Total no	Mean CCT(μm)	Std. deviation	P-value
Males	52	568.48	33.475	0.272
Females	48	563.43	31.178	

TABLE 4: Association of CCT with duration of diabetes.

Duration of diabetes (in years)	CCT		χ^2	p
	<570 μm	\geq 570 μm		
<10 years	30 (50%)	30(50%)	2.192	0.139
\geq 10 years	14 (35%)	26(65%)		

VI. Discussion

Diabetes reduces the activity of $\text{Na}^+\text{K}^+\text{ATPase}$ of the corneal endothelium and this causes the morphological and functional changes of diabetic cornea[11]. Mean CCT of 50 diabetic patients were 574.359 μm and that of non-diabetics were 557.769 μm .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 ($p=0.000$).Most studies and the present study showed that diabetic eyes had increased CCT when compared to non-diabetic subjects[12, 13,14,15].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 [12,14,16]. 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 [17] Intracellular accumulation of sorbitol, which acts as an osmotic agent leads to swelling of the endothelial cells. The Krebs'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.

VII. 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.

REFERENCES

- [1]. L Longanesi, GMCavallini andRToni, Quantitative clinical anatomy of the human cornea in vivo. A morphometric study by ultrasonic pachymetry and computer assisted topographic videokeratoscopy, *Acta Anat* 157(1), 1996, 73-9.
- [2]. JE Sutphin, JChodosh, MR Dana, WCFowler, JS Weiss, PW Turgeon, Examination techniques for the external eye and the cornea, In: Carol LD, Christine Asturo, editor, *External disease of the cornea*, (San Francisco: American Academy of Ophthalmology, 2004) 13-44.
- [3]. GO Waring, WM Bourne, HFEdelhauser, KR Kenyon, The corneal endothelium: normal and pathological structures and functions, *Ophthalmology* 89(6), 1982, 531-90.
- [4]. AJ Bron, RCTripathi, BC Tripathi, The cornea and the sclera. In: Warwick Roger, editor. *Wolff's Anatomy of the eye and orbit*, (London: Chapman and Hall, 1997) 223-78.
- [5]. Fernandez-Vigo Lopez J. Diabetes Ocular LXVIII Ponencia oficial de la Sociedad Espanola de oftalmologia, Barcelona, EDIKA – MED, 1992
- [6]. K Inoue, S Kato, Y Inoue, S Amano, T Oshika, The corneal endothelium and thickness in type 2 diabetes mellitus, *Jpn J Ophthalmol* 46(1), 2002, 65-9.
- [7]. J Siribunkum, PKosirukvongs, ASingalavanija, Corneal abnormalities in diabetes, *J Med Assoc Thai* 84(8), 2001, 1075-83.
- [8]. A Farjo, M McDermott, HK Soong, Corneal anatomy, physiology and wound healing. In: M Yanoff, JS Duker, editors. *Ophthalmology* (St. Louis: Mosby, 2009) 203-8.
- [9]. V Yaylali, SC Kaufman, HW Thompson, Corneal thickness measurement with orbscan topography system and ultrasonic pachymetry, *J Cataract Refract Surg* 23(9), 1997, 1345-50.
- [10]. 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, *Ophthalm Surg* 14, 1983, 750-4.
- [11]. PR Herse, Corneal hydration control in normal and alloxan-induced diabetic rabbits, *Invest Ophthalmol Vis Sci*, 31(11), 1990, 2205-13.
- [12]. 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.
- [13]. 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.
- [14]. AM Roszkowska, CGTringali, PColosi, CASqueri, G Ferreri, Corneal endothelium evaluation in type 1 and type 2 diabetes mellitus, *Ophthalmologica* 213(4), 1999, 258-61.
- [15]. 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.
- [16]. ME Rosenberg, TMTTervo, JJ Immonen, LJ Muller, CGronhagen-Riska, MHVesaluoma, Corneal structure and sensitivity in type 1 diabetes mellitus, *Invest Ophthalmol Vis Sci* 41(10), 2000, 2915-21.
- [17]. 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.