Association Between Central Corneal Thickness and Age-Related Macular Degeneration*

Yaşa Bağlı Makula Dejenereransı ve Santral Kornea Kalınlığı Arasındaki İlişki

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Original Article *Klinik Çalışma*

ABSTRACT

Purpose: To determine any relationship between the development of age-related macular degeneration (AMD) and central corneal thickness (CCT).

Material and Methods: The CCT of patients with neovascular or nonneovascular AMD and of healthy control subjects were measured by using ultrasound corneal pachymetry. ANOVA test was used to compare the CCT between the groups.

Results: The mean age was 71.1 years in the AMD group, and 65.5 years in the control group. There were 100 eyes (100 patients,1 eye from each patient) in the AMD group and 116 eyes (116 patients,1 eye from each patient) in the control group. Half of the 100 eyes with AMD had neovascular AMD and the remaining had nonneovascular AMD. The mean CCT was 550.29±28.58 μ (462-620) in the AMD group as a total and 547.75±33.80 μ (485-650) in the control group ($p>0.05$). The CCT of neovascular AMD eyes was thinner (545.45±25,26 μ) (472-589) than that of both the control group (547.75±33.80 μ) and the nonneovascular AMD group (557.35±31.00 μ) (462-620), but the difference was not statistically significant. The difference between the neovascular and nonneovascular AMD groups was not statistically significant (545.45 vs. 557.35 μ, $p=0.07$).

Conclusion: CCT do not seem to have an association with the development of neovascular or nonneovascular AMD.

Key Words: Corneal thickness, age related macular degeneration.

ÖZ

Amaç: Yaşa bağlı makula dejeneresansı (YBMD) ile santral kornea kalınlığı (SKK) arasındaki muhtemel ilişkiyi değerlendirmek.

Gereç ve Yöntem: YBMD tanısı almış hastalar ve yaşa göre eşleştirilmiş sağlıklı bireylerin ultrason pakimetri kullanılarak SKK ölçümü yapıldı. YBMD grubu sağlıklı grupla SKK'ları açısından ANOVA testi kullanılarak karşılaştırıldı.

Bulgular: YBMD grubunda yaş ortalaması 71.1, kontrol grubunda 65.5 idi. YBMD grubunda 100 göz (100 hasta, herbir hastanın 1 gözü), kontrol grubunda 116 göz (116 hasta,herbir hastanın 1 gözü) çalışmaya dahil edildi. YBMD grubunda 100 gözün yarısında yaş tip diğer yarısında kuru tipte YBMD mevcuttu. Ortalama SKK YBMD grubunda 550.47±28.58 μ (462-620) iken normal grupta 547.75±33.80 μ (485-650) idi ($p>0.05$). SKK yaş tipte YBMD olan gözlerde (545.45±25.26 μ) (472-589) kontrol grubu (547.75±33.80 μ) ve kuru tipteki YBMD olan gözlere (557.35±31.00 μ) (462-620) kıyasla daha ince idi, fakat istatistiksel olarak anlamlı değildi ($p>0.05$). Yaş ve kuru tipte YBMD olan gözlerde iki grup arasındaki farkta istatistiksel olarak anlamlı değildi (545.45 vs. 557.35 μ, $p=0.07$).

Sonuç: SKK, YBMD olgularında hangi tip dejenerasyon gelişeceğini göstermede yardımcı bir faktör değildir.

Anahtar Kelimeler: Santral kornea kalınlığı,yaşa bağlı makula dejeneresansı.

Ret-Vit 2009;17:251-254

Geliş Tarihi : 28/07/2009
Kabul Tarihi : 20/10/2009

Received : June 28, 2009
Accepted : September 20, 2009

* Bu çalışma TOD 2006 Ulusal Oftalmoloji Kongresi'nde sunulmuştur.

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INTRODUCTION

Age-related macular degeneration (AMD) is a very common cause of central vision loss in the elderly people.[1] As the name suggests, it is a condition that tends to become more common as people get older. The cause of AMD is poorly understood, but it is most likely a complex disease in which several risk factors seem to have a potential role.[2] Cigarette smoking, the most consistent risk factor for onset of the disease, and, less consistently, blood pressure, pulse pressure, lipid levels, abdominal obesity, physical activity, dietary fat, sunlight exposure and cataract surgery have been associated with development and progression of AMD.[3]

The knowledge of central corneal thickness (CCT) is of great significance clinically. Patients with primary open-angle glaucoma who had thinner corneas tended to have more severe glaucomatous damage on initial examination by a glaucoma specialist. CCT was the most consistent predictor of degree of glaucomatous damage.[4] In addition, CCT has been associated with several systemic ocular conditions such as aging, diabetes, active Behcet disease, Down syndrome, osteogenesis imperfecta, pregnancy, dry eye, keratoconus, and retinal detachment.[5-11,21,22]

The aim of the present paper was to investigate the CCT values in different types of AMD patients and to check if they differ from the healthy subjects.

MATERIALS AND METHODS

Patients with dry or wet AMD and healthy subjects without AMD were evaluated to be included in the study. A complete ophthalmologic examination was performed to all eyes including visual acuity, slit-lamp examination, and indirect ophthalmoscopy. Exclusion criteria were the use of contact lens, presence of any corneal pathology, glaucoma, hypertensive retinopathy,retinal disease other than AMD or history of previous ophthalmic surgery (except photodynamic therapy for patients with neovascular AMD), smoking, atherosclerosis, collagen tissue disease, refractive pathology other than myopia more than -3 D and presbyopia. An informed consent was obtained from all of the cases. Patients with AMD were divided into wet and dry group and formed the study groups and healthy patients with only refractive problems formed the control group.

The CCT was measured by using ultrasound pachymetry (Advent Pachymeter; Mentor). All of the measurements were performed by the same experienced ophthalmologist and same time of day. Distribution of the patients were unaware of the person who made measurement. Corneal thickness measurements were carried out during the morning hours. to avoid the diurnal variation in corneal thickness and before any corneal invasive examination procedure (such as Goldmann tonometry). Corneal anesthesia was achieved using proparacaine hydrocloride 0.5% (Alcaine, Alcon) applied immediately before measurement. The probe tip of the pachymeter was held perpendicular to the cornea and centered over the pupil. Each patient was asked to blink before CCT measurements to prevent corneal drying. Five concecutive measurements were made at the center of the cornea of each eye. The lowest CCT measurement was used in the statistical analysis because it was thought to most likely reflect a perpendicular placement of the pachymeter probe and therefore to be the most accurate measurement.[5,6,12]

Patients were classified as having AMD on the basis of standard findings by clinical examination, optical coherence tomography and fluorescein angiography. AMD was named as neovascular AMD (wet form) or nonneovascular AMD (dry form) according to the following criteria; Neovascular AMD included both classical and occult membranes of any age. Nonneovascular AMD was defined as the presence of hyperpigmentation or hypopigmentation associated with drusen or geographic atrophy without choroidal neovascularization. The control group included patients who were examined for presbyopia without evidence of drusen or retinal pigmentary changes.

The CCT values of the groups were compared using ANOVA test. P values below 0.05 were considered as statistically significant.

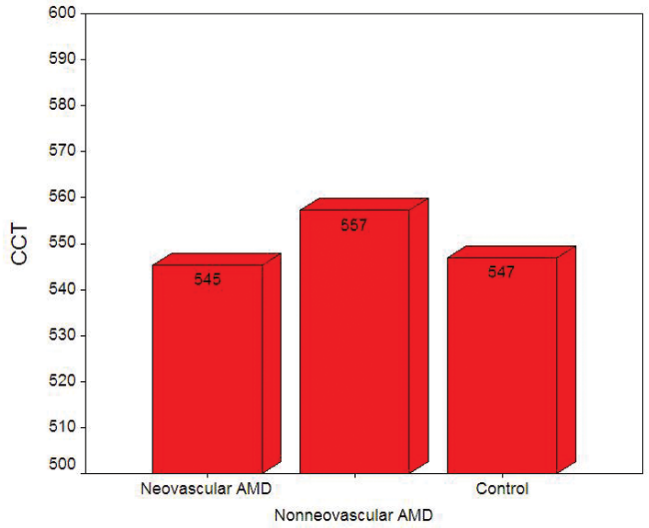
RESULTS

A total of 100 eyes (100 patients,1 eye from each patient) with AMD (AMD group) (50 exudative and 50 nonexudative AMD) and 116 age matched control eyes

Table: The demographic and clinical characteristics of the participants.

Charestericticts	Age (yr; mean±SD)	F/M	Visual acuity (mean±SD)	CCT (μm; mean ±SD)
AMD group	71.17±17	57/43	0.42±0.35	551.29±29.65
Neovascular	72.25±9.51	28/22	0.36±0.33	545.45±25.26
Nonneovascular	70.10±8.72	29/21	0.48±0.36	557.35±31.00
Control Group	68.0±24	66/50	0.85±0.23	547.75±33.80

p=0.18, ANOVA test.



Graphic: Graphical view of the distribution of patients.

(116 patients,1 eye from each patient) (control group) were involved in the study.

Table 1 presents the demographic, and clinical characteritics of the participants. There was no statistically significant difference between the groups with respect to age and gender. The visual acuity of the AMD group (0.42±0.35) was significantly lower than the control group (0.85±0.23), (p=0.00) as expected.

CCT measurements were also similar in two groups; AMD group: 550.4±28.5 (462-620) and control group: 547.7±33.8 (485-650). When we further divided the AMD group into two as neovascular and nonneovascular group, the mean CCT was lower in the neovascular AMD group 545.4±25.2 (472-589) and higher in the nonneovascular AMD group 557.3±31.0 (462-620) than the control group 547.7±33.8 (Grphic). However the differences between the groups were not statistically significant (p=0.18).

DISCUSSION

Corneal thickness could be representing a biomechanical parameter of the eye with several implications. An association between CCT and several clinical conditions has been documented (such as optic nerve drusen, p, tuitary adenoma, pregnancy, osteogenesis imperfecta, glaucoma, diabetes, and retinal detachment).[4-11]

Several ocular risk factors such as cataract extraction, iris color, and refractive errors have been described to be involved in the development of AMD in addition to systemic factors like smoking, atherosclerosis, and genetic factors.[1,2,13-17] The possible pathophysiologic mechanism by which corneal thickness may be implicated in the pathogenesis of choroidal neovascularization in AMD could be the correlation between corneal thickness and corneal elasticity, scleral stiffness, and ocular rigidity.[18] In 1989, Friedman and associates found that increased ocular rigidity may be a significant risk factor in the development of AMD. The possible pathophysiologic mechanism that has been proposed to explain this association was that the sclera in eyes with AMD becomes increasingly rigid and noncompliant, which increase the resistance of venous outflow and decreases the choroidal blood flow. The final result of this cascade is that there is a decompensation of the choroidal venous system at the posterior pole, the Bruch membrane, and the retinal pigment epithelium of the macular area, leading to the development of AMD.[19,20]

In the present study, we failed to demonstrate any association between AMD and CCT. Although CCT measurements were lower in the neovascular AMD group and higher in the nonneovascular AMD group than the control group, the differences were not statistically significant. This finding is also supported by the study by Kymionis et al; however, they have only studied on neovascular AMD patients.[23] On the other hand, the difference may reach to a statistically significant level when the numbers of patients are increased in the study. If so, CCT could be used as a practical indicator in identifying patients at risk of developing different types of AMD. The most important limitation of this study is the small sample size. The results would be more reliable when it is enlarged.

In coclusion, despite the described limitations of the study, our study suggests that CCT do not seem to have a role in the development of neovascular or nonneovascular AMD. Further studies with larger sample size are needed to elucidate a possible correlation of CCT with neovascular AMD.

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