Central Choroidal Thickness in Eyes With Central Serous Chorioretinopathy: The Effect of Disease Activity

Santral Seröz Korioretinopatili Gözlerde Santral Koroidal Kalınlık: Hastalık Aktivitesinin Etkisi*

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ABSTRACT

Purpose: To examine central choroidal thickness in eyes with central serous chorioretinopathy (CSC) and to determine the effect of disease activity to thickness using spectral-domain optical tomography (SD-OCT)

Materials and Methods: Fourteen subjects with CSC in active phase (16 affected eyes and 12 fellow eyes) , 12 subjects with CSC in inactive phase (24 eyes) and 20 subjects (40 eyes) with no retinal or choroidal disease, underwent SD-OCT testing with EDI technique.

Results: The choroidal thickness at the center of the fovea during the active phase in the affected eyes (mean 505.2 μm), in the fellow eyes (mean 447.3 μm) and during the inactive phase of the CSC subjects (mean 399.5 μm) was significantly thickened compared with the control group (mean 287 μm; P <0.05).

Conclusion: Central choroidal thickness is thicker in all CSC subjects than in normal subjects. The activity of the disease may affect the thickness of the choroid.

Key Words: Central serous chorioretinopathy, choroidal thickness, optical coherence tomography.

ÖZ

Amaç: Santral seröz korioretinopatili (SSKR) gözlerde spektral domain optik kohorens tomografi (SD-OKT) kullanarak santral koroidal kalınlığı değerlendirmek ve hastalık aktivitesinin kalınlığa etkisini belirlemek.

Gereç ve Yöntem: Aktif faz SSKR'si olan 14 olgu (16 etkilenen göz ve 12 diğer göz), inaktif SSKR'li 12 olgu (24 göz), herhangi bir retinal ve koroidal hastalığı olmayan 20 olgu (40 göz) SD-OKT'nin EDI (Enhanced Depth Imaging-Arttırılmış Derinlik Görüntüleme) yöntemiyle değerlendirildi.

Bulgular: Santral koroidal kalınlık ölçümleri SSKR'li olgularda aktif fazda etkilenen gözde (ortalama 505.2 μm), diğer gözde (ortalama 447.3 μm) ve inaktif fazdaki olguların gözlerinde (ortalama 399.5 μm) kontrol grubundaki gözlere (ortalama 287 μm) kıyasla anlamlı derecede kalın bulundu (p <0.05).

Sonuç: Santral koroid, SSKR'li tüm gözlerde normal olgulardan daha kalındır. Hastalık aktivitesi koroid kalınlığını etkileyebilir.

Anahtar Kelimeler: Santral seröz korioretinopati, koroidal kalınlık, optik koherens tomografi.

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INTRODUCTION

Central serous chorioretinopathy (CSC) is characterized by serous retinal detachment in the macular area. The disease often resolves spontaneously with a good visual acuity. Fluorescein angiography usually demonstrates dye leakage from the retinal pigment epithelium and subretinal dye pooling. When the detachment spreads into the central macular area, the patient typically develops metamorphopsia, a positive scotoma, and micropsia. The exact underlying mechanism of the disease is unclear.[1-5] Recent indocyanine green angiography studies of patients with CSC have shown hyperpermeability of the choroidal vasculature.[5-7]

Over the last decade optical coherence tomography (OCT) has become an integral tool in the field of ophthalmology. The progression of this new technology, give us the ability to obtain a true, noninvasive "optical biopsy" of the posterior segment.[8]

It is known that, the choroid plays a vital role in the pathophysiology of many diseases affecting the retina, but adequate visualization of the choroid using OCT has not been possible until recently. Improvements to existing spectral-domain OCT (SD-OCT) devices, such as frame averaging, despeckling, and improved image contrast, provide even better definition of intraocular structures such as choroidal tissue.[9,10] Spaide and colleagues[9] described a technique named "enhanced depth imaging" or "EDI" to optimize the parameters of OCT acquisition to facilitate visualization of the full thickness of the choroid. Subsequently, a variety of techniques, including the use of longer imaging wavelengths, have been advanced to promote analysis of the choroidal thickness.[9-11] There are some studies in the literature including choroidal thickness measurements in CSC patients.[12-19]

In this study our aim is to examine the effect of disease activity to central choroidal thickness in eyes of CSC subjects using SD-OCT with EDI technique.

MATERIAL AND METHODS

We prospectively studied fourteen subjects with CSC in active phase (16 affected eyes and 12 fellow eyes) , 12 subjects with CSC in inactive phase (24 eyes) and 20 subjects (40 eyes) with no retinal or choroidal disease.

The study was performed in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants. The diagnosis of CSC was based on the presence of serous macular detachment documented by OCT and leakage from the retinal pigment epithelium on fluorescein angiography and the absence of other chorioretinal disorders that can cause macular exudation such as drusen, pathologic myopia, intraocular inflammation, retinal vasculopathies, angioid streaks, trauma, and hereditary dystrophies.

Cases with active and inactive CSC were included. Active CSC was defined as a serous macular detachment caused by one or several isolated leaks seen on fluorescein angiography at the level of the retinal pigment epithelium and serous macular detachment observed on OCT. Inactive CSC was defined as staining of the retinal pigment epithelium atrophy without leakage and the absence of the fluid on OCT in patients who were diagnosed as CSC before and followed up with or without treatment.

All patients had a complete ophthalmologic examination, including binocular fundus biomicroscopy. Best-corrected visual acuity (BCVA) was measured with Snellen chart. All patients underwent fluorescein angiography using the Zeiss FF 450 (Carl Zeiss Meditec AG, Germany) or the Heidelberg scanning laser ophthalmoscope (Heidelberg Engineering, Heidelberg, Germany). OCT was performed using the SD-OCT with EDI software (Heidelberg Engineering, Heidelberg, Germany) with a standardized scanning protocol. For each eye, a 31-line raster scan centered on the fovea (with tracking on) was performed, with 25 frames averaged to improve the image quality. The EDI technique, with the zero delay line oriented to the choroidal side, was used to optimize choroidal sensitivity and enhance visualization of the full choroidal thickness.

Choroidal thickness at the fovea was measured using the caliper tools of the proprietary software (Heidelberg Eye Explorer) on the OCT machine. The choroidal thickness was measured using a line drawn perpendicularly from the hyperreflective line believed to represent the retinal pigment epithelium to the choroid-sclera junction. The measurements were performed by the same grader who was the only experienced author in the study. To minimize the effect of diurnal variation on choroidal thickness, the OCT scans were done in a time range beginning from 1 PM to 5 PM (Since choroidal tissue is found to be thicker in morning hours in recent studies).[20,21]

Statistical Analysis: All statistical analysis were performed using the software SPSS 15.0 for Windows (SPSS Inc., Chicago, IL, USA). A p value of 0.05 or less was considered as statistically significant; one-way ANOVA was used to analyze the parameters. The results were expressed as mean values with their standard deviations (SD).

RESULTS

The mean age of the active CSC subjects, inactive CSC subjects and control group were 34.25, 38.4 and 35.6 years respectively. There was no difference between the ages of the groups ($p>0.05$).

In the active CSC group, all of the patients complained of reduced central vision in the study eye for one month or less. Ten of the 14 patients described metamorphopsia. Four of the 12 patients in the inactive CSC group complained of visual disturbances despite the absence of fluid on OCT. There was no visual disturbances in the unaffected fellow eyes of active and inactive CSC patients and the fundus examinations of these patients showed normal findings.

Binocular Fundus Examination: A neurosensory macular detachment was detected in all active CSC eyes. Pigment defects of the retinal pigment epithelium were observed in or around the fovea in 11 eyes of the active CSC group and in 10 eyes of the inactive CSC group.

Fluorescein Angiography: Fluorescein angiography revealed only one leaking point (4 subfoveal and 7 juxtafoveal) in 11 eyes of active CSC patients. Remaining five eyes of the active CSC patients showed multifocal leaks (one of them involving foveal center) in angiographic evaluation. All eyes with inactive CSC had some degree of transmission hyperfluorescence corresponding to retinal pigment epithelium atrophy and there was no leakage in these patients.

OCT Findings: All eyes in active CSC group had neurosensory macular detachment involving macular area on OCT. Three of these eyes also had one or more small retinal pigment epithelium detachments. Five eyes of the active CSC group showed extrafoveal neurosensory macular detachment areas. Central foveal thickness was found to be 368.65 ±48.74 μm in active CSC patients.

All eyes with inactive CSC had some degree of retinal pigment epithelium disturbances and eight eyes had small inner segment-outer segment (IS/OS) junction defects on OCT.

Visual Acuity Measurements: The mean visual acuity of the eyes with active CSC, inactive CSC and control groups were, 0.83, 0.98, 1.0 Snellen lines respectively and the difference between groups was not significant (p>0.05). There was no difference between the refractive errors of the groups (p>0.05).

When we evaluate the records of fourteen eyes who were studied as inactive CSC, the resolution of macular detachment occurred with medical treatment (oral acetazolamide and topical non-steroidal anti-inflammatory drugs) in eight eyes, after argon laser photocoagulation in two eyes or after photodynamic therapy (PDT) in two eyes. Argon laser was applied in two patients that have no resolution of the fluid with medical treatment for three months. During the laser 100 mw energy with 100 msn and 100 microns was used on extrafoveal retinal pigment epithelium leaking points. PDT was applied as an half-dose regimen in two patients with subfoveal leakage that have the disease longer than three months. Medical treatment was recommended as a first line treatment for the patients with a disease history shorter than three months. The last evaluations of the treated patients were performed at the third month.

When we compare the central foveal thickness measurements, we found significant difference between the affected eyes of the active CSC subjects and the fellow eyes of active CSC subjects, both eyes of the inactive CSC subjects and the control eyes (p <0.05) (Table 1).

When we evaluate the choroidal thickness measurements, it was found that the choroidal thickness at the center of the fovea during the active phase in the affected 16 eyes (range, 462 to 568 μm; mean 505.2 μm), in the fellow 12 eyes (range, 295 to 515 μm; mean 447.3 μm) and during the inactive phase of the CSC subjects (range, 348 to 500 μm; mean 399.5 μm) was significantly thickened compared with the control group (range, 241 to 313 μm; mean 287 μm; P <0.05) (Table 1, Figure 1, 2, 3 and 4). When we compare the choroidal thickness measurements of active and inactive CSC patients we did not find any significant difference between the affected eyes and the fellow eyes of active CSC subjects, between the affected eyes, the fellow eyes of active CSC subjects and the inactive CSC eyes (p>0.05).

DISCUSSION

In CSC, fluid accumulates below the neurosensory retina and results detachment of the neurosensory retina. Although the mechanism for the development of central serous chorioretinopathy remains unclear, some mechanisms have been proposed about the retinal pigment epithelium, choroid, or Bruch's membrane. It has been proposed to be a weakening of the adherence of the retinal pigment epithelial cells to each other and to Bruch's membrane. This causes the breakdown of the retinal pigment epithelium, and leaking of the serous fluid from the underlying choriocapillaris into the subretinal space.

Table 1: *Central foveal and central choroidal thickness measurements of the patients with active, inactive CSC and controls.*

	Active CSC subjects affected eye	Active CSC subjects fellow eye	Inactive CSC subjects both eyes	Control subjects	p value
Central choroidal thickness (μm) Mean±SD	505.22±48.21	447.28±39.15	399.48 ±50.36	287.18 ±49.23	p<0.05 *
Central foveal thickness (μm) Mean±SD	364.52 ±58.69	202.66 ±27.07	195.36 ±37.12	220,14± 18,13	p<0.05 **

*: When we evaluate the choroidal thickness measurements, we found that the central choroid in the affected eyes and the fellow eyes of the active CSC subjects, in both eyes of the inactive CSC subjects was significantly thicker than the central choroid of the control eyes (p <0.05). When we compare the choroidal thickness measurements of active and inactive CSC patients we did not find any significant difference between the affected eyes and the fellow eyes of active CSC subjects, between the affected eyes, the fellow eyes of active CSC subjects and the inactive CSC eyes (p>0.05).

**: When we compare the central foveal thickness measurements we found statistically significant difference between the affected eyes of the active CSC subjects compared with the fellow eyes of active CSC subjects, both eyes of the inactive CSC subjects and the control eyes (p <0.05).

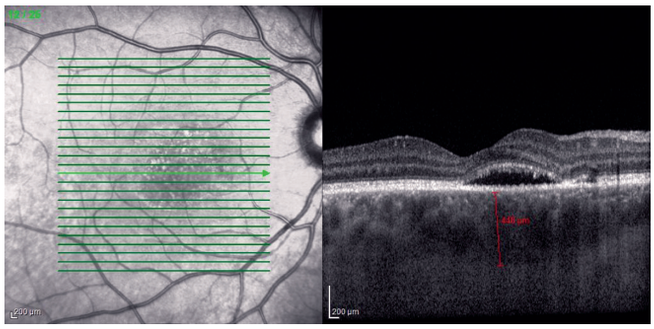

Figure 1: *Central choroidal thickness in the affected eye of the active CSC patient with neurosensory macular detachment on OCT. The choroidal thickness is found to be 448 µm.*

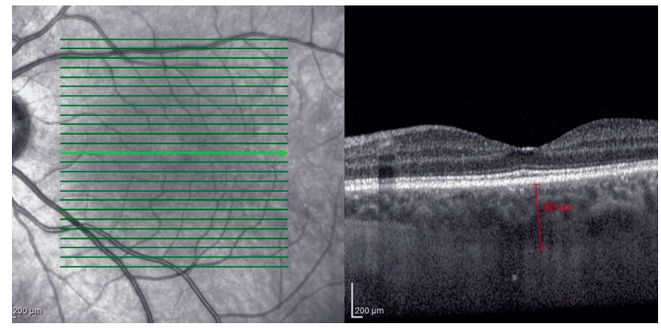

Figure 2: *Central choroidal thickness in the fellow eye of the same CSC patient in figure 1. It is found to be 369 µm.*

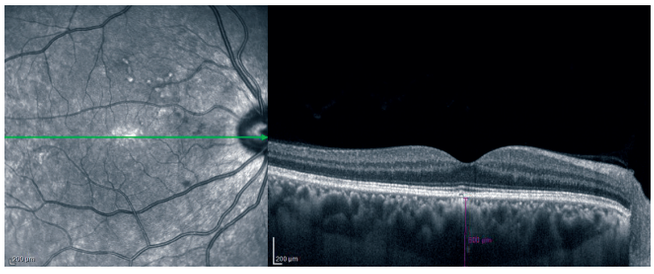

Figure 3: *Central choroidal thickness in the affected eye of the inactive CSC patient. Although there is no fluid on OCT the choroid is stil thick. It is found to be 500 µm.*

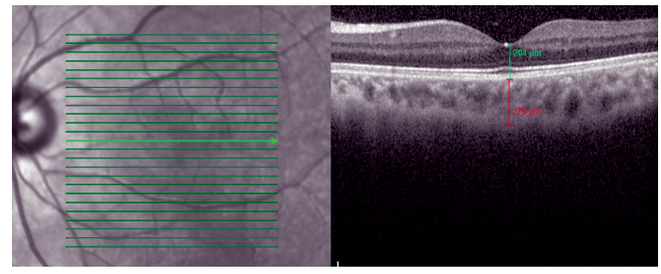

Figure 4: *Central choroidal thickness is 279 µm in an healthy control subject.*

Another proposed mechanism is about the choroidal vasculature. Recent indocyanine green angiography studies of patients with CSC have shown hyperpermeability of the choroidal vasculature.[22-24] CSC-related retinal pigment epithelium detachments often occur over areas of choroidal hyperpermeability.[22] The extravascular fluid present in the choroid may produce a fluid pressure that affects retinal pigment epithelium ion pumps and may allow fluid to pass into the subretinal space creating the serous retinal detachment.[25,26]

Until recently, information regarding choroidal thickness in human eyes was based primarily on histologic results, which do not necessarily reflect the true measurements of this dynamic tissue. With advancements in OCT image processing software, more refined details of the posterior segment can be evaluated in vivo. Recent OCT imaging technique has revealed accurate findings of choroidal thickness in known diseases such as CSC.

There are several studies showing the choroidal thickness in CSC. Kim et al.,[16] evaluated the choroidal thickness among patients with healthy eyes, early age-related maculopathy, neovascular age-related macular degeneration, CSC and polypoidal choroidal vasculopathy. They found that the choroid was thicker in polypoidal choroidal vasculopathy (319.92±68.66 µm) and CSC (367.81±105.56 µm) patients than in controls (241.97±66.37 µm) and age-related maculopathy patients (186.62±64.02 µm).

Another study by Kim et al.,14 showed that, increased choroidal thickness in patients with unilateral CSC was noted not only in the affected eyes, but also in the unaffected fellow eyes. ICGA revealed choroidal vascular hyperpermeability in 93.3% of eyes with CSC and in 73.3% unaffected fellow eyes. Choroidal vascular dilation was detected in 70.0% of eyes with CSC and in 60.0% unaffected fellow eyes.

Maruko et al.,[15] examined sixty-six consecutive Japanese patients with unilateral CSC. The subfoveal choroid in symptomatic eyes was significantly thicker than that in fellow eye.

Jirarattanasopa et al.,[27] evaluated 34 patients with CSC (44 eyes) and 17 volunteer subjects (17 normal eyes). Mean whole macular choroidal thickness in eyes with CSC (total, 329.3±83.0 µm) was greater than that in normal eyes. There was no difference between the choroidal thickness of classic CSC, chronic CSC and multifocal posterior pigment epitheliopathy. In unilateral cases, mean whole macular choroidal thickness was greater in eyes with unilateral CSC than in unaffected fellow eyes.

In our study, choroidal thickness measurements in active and inactive phase of CSC across the macula demonstrated a thick choroid. Although the choroid was thinner in inactive CSC patients who received different types of treatments and with no fluid on OCT than the active CSC patients; we did not find any significant difference. This finding may be due to the disturbance of the choroid in CSC subjects both in active and inactive phase and the continuity of the pathology after treatment. Increased choroidal thickness in patients with unilateral CSC was noted not only in the affected eyes, but also in the unaffected fellow eyes.

As a conclusion, the results of this study suggest that CSC might be an essentially bilateral disorder. Central choroidal thickness is thicker in all CSC subjects than in normal subjects and the activity of the disease may increase the thickness.

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