Comparative Study between Central Subfoveal Choroidal Thickness in Diabetic Retinopathy and Control by Spectral Domain Optical Coherent Tomography

Noura Khalid Al Qassimi¹ and Nagla Hassan Aly²*

¹German Board, Ophthalmology Consultant at Sheik Khalifa Medical City, Abu Dhabi, UAE
²Assistant Professor at Memorial Institute for Ophthalmic Researches (MIOR), Giza Egypt

*Corresponding Author: Nagla Hassan Aly, Assistant Professor at Memorial Institute for Ophthalmic Researches (MIOR), Giza Egypt.

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Abstract

Purpose: To compare subfoveal choroidal thickness (SCT) measurements by commercially available spectral-domain optical coherence tomography instruments between healthy eyes and moderate non-proliferative diabetic retinopathy without macular edema.

Patients and Methods: 80 eyes of patients whom ages ranged from 45 - 55 years both sex, were subjected to full ophthalmological examination including slit lamp biomicroscopy and FA for staging of the Disease (moderate NPDR without macular edema).

• Best corrected visual acuity not less 20/40.
• IOP less than 21.
• Choroidal thickness (CT) was measured from the outer edge of the hyperreflective retinal pigment epithelium to the inner sclera at subfoveal region.

Accordingly the patients were divided into 2 groups:

• Group (1) 40 eyes of normal control.
• Group (2) 40 eyes with moderate non-proliferative diabetic retinopathy type 2 where The mean central subfoveal choroidal thickness was measured manually by SD OCT (Topcon–seven lines protocol).

Results: Subfoveal choroidal thickness showed significant decrease in moderate NPDR compared to control group.

Conclusion: Mean central subfoveal choroidal thickness is decreased in moderate non-proliferative diabetic retinopathy than normal control using spectral domain OCT.

Keywords: Correlation; Normal; OCT; Early Diabetic Retinopathy; Subfoveal Choroidal Thickness

Abbreviations

DM: Diabetes Mellitus; NPDR: Non-Proliferative Diabetic Retinopathy; MNPDR: Moderate non-Proliferative Diabetic Retinopathy; EDI: Enhanced Depth Imaging; OCT: Optical Coherent Tomography; SD: Spectral Domain; CT: Choroidal Thickness; SCT: Subfoveal Choroidal Thickness

Introduction

Retina is a highly functioning organ with increased metabolic demand, which considered the highest blood flow rate among the other vital organs in the body [1]. The choroidal vasculature, especially the choriocapillaris, provides oxygen and nutrients to the outer retina and is responsible for maintaining the highly metabolically active retinal layers, and as the choroid is one of two blood suppliers to the retina and its primary role is to provide approximately 85% of the blood supply to the retina including all the photoreceptors and the...
entire retinal pigment epithelium (RPE) [2] (Figure 1). Photoreceptors promptly degenerate if choroidal circulation or innervations to the choroid is disrupted [3,4].

Figure 1: Choroid provide approximately 85% of the blood supply to the retina including all the photoreceptors and the entire retinal pigment epithelium.

To ensure a flawless oxygen supply to the retina, the choroidal blood supply must maintain a very high oxygen tension, which is achieved through high blood flow resulting in an arterial/venous oxygen tension difference of only 3% [5]. With the introduction of enhanced depth imaging (EDI) mode of optical coherence tomography (OCT), visualization of this important structure has become possible, which has increased our understanding of the choroidal changes in certain diseases such as central serous chorioretinopathy, age-related macular degeneration, polypoidal choroidal vasculopathy, choroidal neovascularization, and glaucoma [6-9]. The thickness of the choroid in some of these conditions represents a potential follow-up parameter in the course of the diseases [10].

Diabetes mellitus (DM) might cause choriocapillaris loss, increased tortuosity, narrowing of vessels, and sinus-like structure formation between choroidal lobules [11]. The choroidal status in DM has recently gained attention after the role of the choroid in certain ocular diseases was assessed. However, the results are conflicting in different literatures as some showed thinning of the choroid [12], others showed thickening of the choroid [13], or sometimes there is no changes affecting the choroidal layer thickness [14].

Choroid is a vascular structure that has rich neuronal innervations, thus neurogenic mechanisms might be strongly involved in choroidal blood flow regulation [15]. We hypothesized that Diabetic retinopathy might cause choroidal thickness (CT) changes in diabetes mellitus that increased if associated with neuropathy. For this purpose, we tried to compare the CT values in controls and patients also in patients with moderate NPDR without macular edema [16].

Spectral-domain optical coherence tomography (SD-OCT) technology improves resolution, compared with time-domain OCT. SD-OCT scans allow seeing even small retinal details of photoreceptor layer, such as the inner segment/outer segment (IS/OS) junction. Recently, a new approach to improve the depth imaging by OCT, termed enhanced depth imaging (EDI) OCT, has been shown to be able to reliably image the fullthickness of the choroid. The net effect of this practice is that the sensitivity of the imaging in deeper layers of tissue is increased. In this fashion OCT may represent a useful approach to investigate, the choroidal changes in eyes with diabetic retinopathy [17].

Patients and Methods

80 eyes of 60 patients whom ages ranged from 45 - 55 years, 35 males and 25 females, were subjected to ophthalmological examination.
The study was approved by the ethical committee of the scientific research. Subjects were given full explanations about the purpose of the study and its consequences and all patients were informed about the steps of the examinations and investigations and accordingly the eyes were divided into 2 groups:

- Group (1) 40 eyes of control.
- Group (2) 40 eyes of moderate non-proliferative diabetic retinopathy type 2.

The mean central subfoveal choroidal thickness was measured manually by OCT (vertical seven lines protocol) done by OCT topcon (3D SD OCT 2000 FA +).

All Patients were examined and divided into 2 groups where group (1) included the control and group (2) including MNPDR with that

**Inclusion Criteria:**
- Best corrected visual acuity not less 20/40.
- IOP less than 21.
- Slit lamp biomicroscopy for staging of the disease detecting moderate NPDR type 2 where the fundus showed presence of scattered hemorrhages, microaneurysms, and hard exudates without macular edema.
- Fluorescein angiography showed microaneurysms appear as hyperfluorescent spots in early phases of the angiogram and typically leaking or not leaking in the later phases of the test without macular edema.
- OCT determined the thickness of the retina and exclude any macular edema followed by manual choroidal thickness through 7 lines protocol.

**Exclusion Criteria:**
- High refractive error > +3.00D or < −7.00D
- Lens opacity or previous intraocular surgery.
- Diabetic macular edema, Proliferative DR, Laser photocoagulation and VEGF therapy, all those were excluded in the study.
- Glaucoma, age related macular degeneration, cystoid macular edema, choroidal neovascularization (CNV), vitreoretinal diseases (i.e., vitreomacular traction syndrome and epiretinal membranes and any other ocular or systemic diseases.

The choroid was measured by 7 vertical lines protocol by Topcon OCT SD on the fovea with choroidal enhancement and the choroidal thickness was measured manually subfoveal from the outer portion of the hyperreflective line corresponding to the RPE to the hyporeflective line or margin corresponding to the sclerochoroidal interface (Figure 2). These measurements were made at the subfoveal choroid and the values of the measurements between normal and MNPDR were compared and statistical analyses were performed by T test to evaluate the relationships between subfoveal choroidal thickness in group (1) control (Figure 3) and in group (2) moderate NPDR (Figure 4).

**Figure 2:** Hyperreflective line corresponding to the RPE to the hyporeflective line or margin corresponding to the sclerochoroidal interface.
Correlation between central choroidal thickness at the fovea in diabetic group and control group was analyzed using the T test and the chosen level of statistical results were significant when p value below 0.05.

**Results**

80 eyes of 60 patients whom ages ranged from 45 - 55 years, 35 males and 25 females, were subjected to full ophthalmological examination.

Choroidal thickness was measured subfoveal from the outer portion of the hyperreflective line corresponding to the RPE to the hyporeflective line or margin corresponding to the sclerochoroidal interface.

Mean SCT in group (1) control group was 331.42 μm and SD is 16.45 in 40 normal eyes (Figure 3).

![Figure 3: Subfoveal Choroidal thickness in group (1) control.](image)

Mean SCT in group (2) 273.28 μm and SD is 13.79 μm in 40 diabetic eyes type 2 (Moderate NPDR) (Figure 4).

![Figure 4: Subfoveal Choroidal thickness decreased in group (2) moderate non-proliferative diabetic retinopathy.](image)

The mean subfoveal choroidal thickness 273.28 μm was significantly reduced in diabetic group compared with the mean subfoveal choroidal thickness in control group 331.42 μm with P < 0.001 (Figure 5).
Discussion

Diabetic retinopathy is due to the breakdown of retinal vasculature integrity and hemodynamic abnormalities, choroidal thickness is measured as the distance between the outer aspect of the hyperreflective layer correspondent to the RPE/Bruch membrane complex and the choroidal-scleral interface. The reproducibility of the total choroidal thickness measurement has been reported, in normal aging eyes, or pathological conditions.

The choroidal changes in diabetic retinopathy have been studied and diverse results have been published. Some studies reported that no difference in central CT between non-proliferative retinopathy and healthy subjects, some with decrease in proliferative retinopathy and diabetic macular edema (DME). Similarly, in a multicenter trial, Esmaeelpour M., et al. have found thinner choroid in DME patients than in healthy volunteers [14].

Querques., et al. have stated that although the diabetic groups in their study (no retinopathy, non-proliferative retinopathy with and without macular edema) have significantly thinner choroids than the controls, there is no difference between the diabetic groups [18]. Vujosevic., et al. have reported no difference between controls and diabetes patients, and DME does not influence the CT [19], however Beijing Eye Study of Xu., et al. have found that the subfoveal CT is thicker in diabetic patients but is not related to the severity of the retinopathy [20].

Regarding subfoveal CT in diabetic patients the literature search did not reach a conclusion about CT and its relationship with the severity of the retinopathy. These controversial results might be attributed to several factors. First, most of the studies are retrospective in nature and include patients who have been treated with photocoagulation or anti-vascular endothelial growth factor (anti-VEGF) agents excluding our study, some studies pointed out this issue and stated that the presence of treated patients might be a limitation of their study. Our study population had the advantage of being untreated, since it is known that photocoagulation or anti-VEGF treatment causes thinning of CT [21].

We used manual measurements in CT, The 7-line raster measurements protocol in our study reflected the whole measurements, however, other published studies showed that single horizontal line scan measurements can represent the subfoveal choroid thickness successfully.
Our findings are consistent with circulatory studies showing a decreased pulsatile ocular blood flow in diabetic patients. In fact, the dropout of the choriocapillaris could increase vascular resistance, resulting in decreased blood flow in the choriocapillaris [22].

Previous studies demonstrated that a decreased choroidal blood flow and decreased choroidal thickness at the fovea, probably due to the dropout of the choriocapillaris (and determining increased vascular resistance), may cause retinal hypoxia, that may occur before the clinical manifestations of diabetic retinopathy. Similarly, in our study the subfoveal choroidal thickness value obtained in the NPDR group was lower than that in the age-matched control group [23].

Recently choroidal thickness change was found in type 2 diabetic eyes, irrelevant of the presence of retinopathy. Type 2 diabetes is often accompanied by systemic pathologies such as high BP and high lipid levels which are known to have adverse effects on the retina. Hence, the thinning found in type 2 diabetic eyes could represent a cumulative effect of the glycemic disease component and other accompanying factors.

**Conclusion**

Mean central subfoveal choroidal thickness were decreased in moderate non-proliferative diabetic retinopathy than normal control measured by SD OCT.

**Bibliography**


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