Choroidal Thickness in Behcet’s Uveitis: An Enhanced Depth Imaging-Optical Coherence Tomography and Its Association With Angiographic Changes

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PURPOSE. To evaluate the change in subfoveal choroidal thickness between active and quiescent phases of Behcet’s posterior uveitis and compare this with the healthy population using enhanced depth imaging optical coherence tomography (EDI-OCT).

METHODS. Thirty eyes from 30 patients with Behcet’s posterior uveitis (mean age, 47.03 ± 11.01 years) were retrospectively enrolled in the study. Their subfoveal choroidal thickness was measured using EDI-OCT in the active and quiescent phases of Behcet’s uveitis, and compared with the age, sex, and spherical equivalent–matched healthy population. Changes in retinal vascular leakage on fluorescein angiography (FA) were correlated with the changes in subfoveal choroidal thickness.

RESULTS. Mean subfoveal choroidal thickness in the acute phase of Behcet’s uveitis was significantly greater than that in the quiescent phase (358.77 ± 155.59 μm versus 356.72 ± 141.09 μm; P = 0.004). Subfoveal choroidal thickness in the quiescent phase was also significantly greater than that of the healthy population (259.96 ± 65.16 μm; P < 0.0001). There was a statistically significant association between the change in subfoveal choroidal thickness and the change in vascular leakage revealed by FA (ρ = 0.381, P = 0.046). Subfoveal choroidal thickness in the uninvolved fellow eyes of patients with unilateral Behcet’s uveitis was also evaluated and it was significantly greater than that of the healthy population (n = 13 eyes; P = 0.001)

CONCLUSIONS. This study found choroidal thickening during the active phase of Behcet’s posterior uveitis. Subfoveal choroidal thickness during the quiescent phase was also significantly greater than in normal eyes. The degree of reduction in choroidal thickening was significantly correlated with improvement in retinal vascular leakage as revealed by FA.

Keywords: Behcet’s uveitis, choroidal thickness, choroid, fluorescein angiography

Behcet’s disease is a chronic, recurrent, inflammatory systemic occlusive vasculitis affecting both arteries and veins in all organs.1 Its ocular involvement occurs in approximately 70% to 90% of patients in the form of anterior uveitis, posterior uveitis, optic neuritis, and retinal vasculitis. Choroidal involvement in Behcet’s uveitis has been previously implicated from histopathologic studies, which reported a diffuse and focal infiltration of the choroid with inflammatory cells.2–4 Using fluorescein angiography (FA) and indocyanine green angiography (ICGA), several in vivo studies have implicated choroidal involvement in Behcet’s uveitis. In addition, involvement of choroidal thickening in Behcet’s uveitis has been shown using A-scan echography.5

Enhanced depth imaging optical coherence tomography (EDI-OCT) has recently become an excellent tool to visualize choroidal layers in living human eyes. Its use in measuring choroidal thickness has been widely adopted for investigating the in vivo anatomy of choroids. Using this method, the characteristics of the choroid in normal and in certain pathologic states (such as high myopia, dome-shaped macula, macula hole, AMD, and Vogt-Koyanagi-Harada syndrome) have been described.6–13 However, EDI-OCT studies of cross-sectional in vivo imaging in Behcet’s uveitis have so far not been reported.

Investigating the in vivo choroidal changes in Behcet’s uveitis has potential significance for understanding its pathophysiology. Although previous histological studies have shown choroidal involvement in Behcet’s uveitis, processing could have at least partially destroyed retinal and choroidal tissue, so the information obtained from histologic specimens might not exactly reflect the in vivo situation. ICGA is useful in visualizing the choroidal vasculature, but it is invasive and difficult to perform repeatedly during the patient’s follow-up. Furthermore, it does not provide sufficient information regarding cross-sectional imaging of the choroid. A-scan can characterize the choroid in a noninvasive manner, but lacks detailed information due to its low resolution and difficulty in differentiating the choroidal layer from the surrounding tissue.
In this retrospective study, we evaluated the in vivo subfoveal choroidal thickness profile of eyes with acute exacerbation of Behcet's uveitis using EDI-OCT to identify any significant differences between the active and quiescent phases of Behcet's uveitis, and to compare these values with that from the age-matched, sex-matched, spherical equivalent-matched healthy population. Furthermore, we assessed the correlation between changes in retinal vascular leakage as revealed by fluorescein angiographic findings and changes in subfoveal choroidal thickness.

METHODS

Patients

The medical records of 110 eyes from 77 patients with Behcet's uveitis who were examined at the Behcet's Uveitis Clinic of Yonsei University Severance Hospital from March 2009 to December 2011 were initially reviewed in this retrospective study. To be included in the study, patients meeting the diagnostic criteria of the International Study Group for Behcet's disease were first identified. Based on the Standardization of Uveitis Nomenclature (SUN) working group classification, patients with evidence of inflammation involving posterior segment (posterior uveitis) were included in the study, and those with anterior uveitis only were excluded. Evidence of posterior involvement of uveitis was documented by the presence of inflammatory cells in the vitreous and/or retinitis, or retinal vasculitis manifesting as perivascular sheathing, vascular leakage, or occlusion on fluorescein angiograms. Patients who had experienced acute recurrent attacks of uveitis after being in the quiescent phase for more than 3 months were identified. Acute attack of uveitis was defined as an episode characterized by sudden onset and limited duration of less than 3 months. Thus, patients whose posterior uveitis had resolved within 3 months or less in duration and whose EDI-OCT and FA were available at the time of acute attack and at least 3 months after resolution of acute exacerbation were enrolled in the study. According to the SUN working group definition, acute exacerbation of active posterior uveitis is defined as eyes with worsening inflammation in the posterior segment with at least one of the following: a two-step increase in vitreous haze score with or without anterior chamber cell involvement, or eyes with angiographic evidence of increased vascular leakage accompanied by visual loss of more than three lines in Snellen visual acuity. An increase in vascular leakage was defined based on the grading system used previously in other studies, and it was determined based on the agreement reached by two independent observers (MK and HJK). Quiescent phase was defined as eyes in an inactive phase of disease for more than 3 months after resolution of acute inflammation, without showing any signs of worsening uveitis, which includes development of retinitis, vasculitis, papillitis, macular edema, and retinal hemorrhage. In addition, subfoveal choroidal thickness of the uninvolved fellow eyes of patients with unilateral Behcet's uveitis was also evaluated and compared to the involved eye in a pairwise manner.

Exclusion criteria were as follows: eyes with spherical equivalent refractive error of more than ±3.0 diopters (n = 5), visually significant cataract or media opacity obscuring the precise visualization of choroidal layers (n = 4), end-stage uveitis with severe chorioretinal atrophy (n = 4), eyes with AMD (n = 2), polypoidal choroidal vasculopathy (n = 1), central serous chorioretinopathy (n = 3), diabetic retinopathy and other concurrent ocular disease (n = 3), or history of intraocular surgery, including cataract, glaucoma, or vitrectomy within 1 year (n = 6). Patients who had received refractive surgeries were also excluded (n = 2). To eliminate the potential influence of previous treatment with intravitreal injections with triamcinolone or antivascular endothelial growth factor agents on the measurement of choroidal thickness, eyes that had received intravitreal injections within the past year were excluded (n = 4).

For the control group, an age-matched, sex-matched, spherical equivalent-matched healthy group was obtained from our database, which included 190 eyes from 190 healthy subjects. Eyes with no ocular disease and no systemic disease, such as diabetes and hypertension, were recruited as control subjects. Subjects were required to have a best-corrected visual acuity of 20/25 or better and spherical equivalent refractive error of less than ±3.0 diopters. Subjects whose acquired OCT images were of poor quality, including an indistinct choriocapillaroid interface, were excluded.

This study was approved by the Institutional Review Boards at the Yonsei University Medical Center (approval number: 4-2011-0894) and was performed in accordance with the tenets of the Declaration of Helsinki. Informed consent was obtained from the subjects after explanation of the nature and possible consequences of the study.

Data Collection

Demographic and clinical information included age at diagnosis of Behcet’s uveitis, sex, duration of Behcet’s uveitis, spherical equivalent of refractive error, and duration of interval between active and quiescent phases of posterior uveitis. All patients were examined by the same uveitis specialist (SCL). All patients underwent a complete ophthalmic examination including slit lamp biomicroscopic examination, measurement of best-corrected Snellen visual acuity, IOP measurement, dilated fundus examination with indirect ophthalmoscope, and fluorescein angiography (HRA 2; Heidelberg Engineering, Heidelberg, Germany). The SUN group grading scheme was used for assessment of inflammation in the anterior chamber and in the vitreous. The spherical equivalent of refractive error was measured by autorefractometry (RK-3; Canon, Tokyo, Japan). All patients underwent spectral domain OCT examination (Spectralis; Heidelberg Engineering).

Image Acquisition Method

Spectral-Domain OCT. The method of obtaining EDI-OCT has been described previously. Briefly, a spectral-domain OCT was placed close enough to the eye to obtain an image. The EDI option allowed the choriretinal interface to be placed adjacent to the zero delay, and an upright image of the retina and choroid was obtained. Spectralis OCT (Heidelberg Engineering) with software version 5.3 was used. The Spectralis OCT contained an 870-nm wavelength superluminescent diode and was capable of obtaining 40,000 A-scans per second with an axial resolution of 7 μm and transversal resolution of 14 μm. Two high-quality horizontal and two vertical line scans passing through the fovea were obtained within a 5 × 30-degree area at the fovea, in which 100 scans were averaged for each section. The signal-to-noise ratio was maximized using the automatic real-time averaging mode to ensure high-quality images. A single drop of Mydrin-P (tropicamide 5 mg/mL and phenylephrine 5 mg/mL; Santen Pharmaceuticals, Osaka, Japan) was administered in each eye at least 30 minutes before EDI-OCT to ensure the eye was dilated. Choroidal thickness was defined as the distance from the outer border of the hyperreflective line corresponding to the RPE perpendicular to the choriocapillaroid interface. Using digital calipers provided by the Heidelberg Spectralis OCT.
software, choroidal thickness was measured at the subfoveal region. A magnification of at least 100% to 200% was used to place the measurement line precisely at the outermost RPE layer and at the choriocapillary interface. Two independent observers (MK and HJK) measured subfoveal choroidal thickness, and these measurements were averaged for analysis. The observers were masked to each other’s measurements and allowed to adjust the contrast of the image to better delineate choroidal boundaries. To prevent bias, a sheet of paper was used to cover the OCT image from the RPE and above so that OCT readers were not allowed to observe the retina from the RPE. Thus, any subretinal or intraretinal fluid attributable to acute exacerbation of Behcet’s posterior uveitis could not be seen by the observers. Improvement in subfoveal choroidal thickness was assessed by the reduction ratio, calculated as (subfoveal choroidal thickness in active phase – subfoveal choroidal thickness in quiescent phase) / subfoveal choroidal thickness in active phase × 100%.

FA. FA images were obtained with a confocal scanning laser ophthalmoscope (HRA 2; Heidelberg Engineering) after intravenous injection with 5 ml of 20% sodium fluorescein. For assessment of the severity of the retinal vascular leakage, a grading system similar to that used previously by others was adopted.15,16 Leakage was classified as none if there was no sign of any vascular staining or leakage (grade 0), mild (grade 1) when there was staining of vessels with minimal leakage, moderate (grade 2) if more intense leakage was observed with a distinct vascular margin, and severe (grade 3) if there was even greater leakage with blurring of the large vessel margins. For the evaluation of the change in vascular leakage during follow-up, the severity of the retinal vascular leakage was graded during follow-up in both active phase and quiescent phase. Experienced retinal specialists who were blind to each other’s readings and the relevant clinical characteristics of the patients evaluated all angiograms independently (MK and HJK). Any disagreement on the FA findings was resolved by open adjudication.

Statistical Analysis

Results are presented as mean ± SD. The Shapiro-Wilk test was used to check for the normal distribution of data, including choroidal thickness in all eyes. Demographic and clinical characteristics, including sex, age, disease duration, and spherical equivalent refractive error, were analyzed using Pearson's chi-square test (Fisher's exact test when expected cell counts were less than five), Student’s t-test, or the Mann-Whitney U test between groups. Comparisons of the choroidal thickness between active and quiescent phases were performed using the Wilcoxon signed-rank test. For evaluation of the association between change in subfoveal choroidal thickness and angiographic vascular leakage, Spearman’s coefficient of correlation was calculated. For evaluation of intraobserver and interobserver agreement, intraclass correlation coefficient (ICC) was calculated. P values less than 0.05 were considered statistically significant. SPSS 18.0 software was used for statistical analysis (SPSS Inc., Chicago, IL).

RESULTS

Thirty eyes from 30 patients with bilateral Behcet’s posterior uveitis (mean age, 47.03 ± 11.01 years; mean spherical equivalent, −0.952 ± 1.097 diopters) were randomly selected and enrolled in the study involving choroidal imaging in active and quiescent phases using EDI-OCT. For the control group, 30 eyes from 30 healthy subjects with age, sex, and spherical equivalent values matching those of the uveitic eyes were included in the study (Table 1). There were no significant differences in age, spherical equivalents, and sex between the two groups. The mean duration of Behcet’s uveitis was 6.43 ± 3.27 years.

In the acute phase of Behcet’s uveitis, mean subfoveal choroidal thickness was 398.77 ± 157.39 μm, which was significantly thicker than that in the quiescent phase (356.72 ± 141.09 μm; Wilcoxon signed-rank test, P = 0.004). There was an average of 15.3% reduction in subfoveal choroidal thickness in the quiescent phase (Table 2). Figures 1 and 2 show representative eyes with Behcet’s posterior uveitis during acute active and quiescent phases, demonstrating increased subfoveal choroidal thickness and prominent vascular leakage during the active phase, which decreased significantly after resolution of acute exacerbation of posterior uveitis. The mean interval between the acute active and quiescent phases in which EDI-OCT images were obtained was 7.04 ± 3.74 months. Choroidal thickness in the quiescent phase was also significantly greater than that of the age-matched, sex-matched, spherical equivalent–matched healthy population (356.72 ± 141.09 μm versus 259.96 ± 65.16 μm; Mann-Whitney U test, P < 0.0001).

Subfoveal choroidal thickness of the unaffected fellow eyes of 13 patients with unilateral Behcet’s uveitis was also evaluated and compared with the involved eye. Subfoveal choroidal thickness in the fellow eyes without overt signs of active Behcet’s uveitis was not significantly different from that in the uveitic eye (P = 0.136), but it was found to be greater than that of the age-matched, sex-matched, spherical equivalent–matched normal eyes (P = 0.001) (Table 3).

Between the two independent observers, a good agreement of subfoveal choroidal thickness measurement was noted with an ICC of 0.982 and intraobserver agreement with an ICC of 0.989 also indicates excellent agreement within observer. All patients received adequate systemic treatment with oral steroids and immunosuppressive agents, such as cyclosporin A, methotrexate, and azathioprine, during the acute phase of posterior uveitis. The subfoveal choroidal thickness did not have a statistically significant correlation with either the duration of uveitis or severity of anterior and posterior inflammation (P > 0.05, data not shown).

DISCUSSION

To our knowledge, this is the first in vivo study to demonstrate choroidal thickening in Behcet’s posterior uveitis using EDI-OCT. We found more subfoveal choroidal thickening during the active phase of Behcet’s uveitis, compared with the quiescent phase in the same eyes. In comparison with the age-matched, sex-matched, and spherical equivalent–matched group, subfoveal choroidal thickness in the quiescent phase was also significantly greater.

The exact pathophysiological mechanism of Behcet’s uveitis is still not known, but we speculate that increased choroidal thickening during the active phase of Behcet’s posterior uveitis could be attributed to several possible mechanisms based on findings from previous histologic studies and imaging studies using FA/ICGA. Obliterative and necrotizing vasculitis is a characteristic histopathological finding in eyes with Behcet’s uveitis. Previously, histopathological studies have demonstrated a diffuse and focal infiltration of the choroid with inflammatory cells such as CD4+ T cells, and macrophag-
analysis was performed for those parameters).

**TABLE 1.** Subfoveal Choroidal Thickness During Active and Quiescent Phases (n = 30 Eyes) in Behcet’s Posterior Uveitis Compared With Age-Matched, Sex-Matched, Spherical Equivalent–Matched Normal Eyes

<table>
<thead>
<tr>
<th>Patients</th>
<th>Active Phase</th>
<th>Quiescent Phase</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n, eyes</td>
<td>30</td>
<td>30</td>
<td></td>
<td>—</td>
</tr>
<tr>
<td>Age, y, mean ± SD</td>
<td>47.03 ± 11.01</td>
<td>47.20 ± 10.72</td>
<td>0.955*</td>
<td></td>
</tr>
<tr>
<td>Sex, male:female</td>
<td>21:9</td>
<td>21:9</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Disease duration, y, mean ± SD</td>
<td>6.43 ± 3.27</td>
<td>NA</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Interval between active and quiescent phase, mo</td>
<td>7.04 ± 3.74</td>
<td>NA</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Spherical equivalent refractive error, diopters, mean ± SD</td>
<td>−0.952 ± 1.097</td>
<td>−0.990 ± 1.146</td>
<td>0.911†</td>
<td></td>
</tr>
<tr>
<td>Subfoveal choroidal thickness, μm</td>
<td>Mean ± SD</td>
<td>Median (range)</td>
<td>P value, active vs. quiescent</td>
<td>P value, active vs. normal</td>
</tr>
<tr>
<td></td>
<td>398.77 ± 157.39</td>
<td>345 (196–756)</td>
<td>0.004‡</td>
<td>&lt;0.0001†</td>
</tr>
<tr>
<td></td>
<td>356.72 ± 141.09</td>
<td>325 (184–715)</td>
<td>266 (129–388)</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>259.96 ± 65.16</td>
<td>65.16</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

Dashes in the P value column indicate that there aren’t any P values associated with each parameter listed in the first column (no statistical analysis was performed for those parameters).

* Independent Student’s t test.
† Mann-Whitney U test.
‡ Wilcoxon Signed-Rank test.

Conventional imaging studies using FA and ICGA in active Behcet’s uveitis have shown various clinical features. Choroidal hyperfluorescence, dye leakage, or choroidal vessel wall staining may be attributable to leukocyte infiltration in choroidal vascular inflammation. Hypofluorescence and choroidal filling defect could be due to accumulation of exudative materials within the stroma, edema and fibrosis, or choroidal vascular obstruction. Also, increased blood flow and vascular resistance during the active phase of inflammation and an association between ocular perfusion pressure and choroidal thickening. Consequently, increased accumulation of exudates arising from altered ocular blood flow due to choroidal vessel inflammation may also contribute to choroidal thickening. We hypothesize that the suggested pathophysiologic changes in the choroid as revealed by histopathology and imaging studies collectively may account for the choroidal thickening observed in our study.

Subfoveal choroidal thickness in the quiescent phase was also significantly greater than that of the age-matched, sex-matched, spherical equivalent–matched healthy population. We speculate that this might be due to subclinical inflammatory activity of the choroid during the quiescent phase, which could exacerbate, leading to an acute recurrent attack of uveitis. Whether choroidal thickening in the quiescent phase is temporary or permanent remains to be investigated, but our results nonetheless suggest that EDI-OCT may be useful in evaluating subclinical choroidal involvement even during the quiescent phases of Behcet’s posterior uveitis. Identifying patients with subclinical disease before the development of overt uveitis may enable preventive measures to be applied at the subclinical stage. Furthermore, because choroidal inflammation in the early phase is thought to have a favorable response to therapy, early identification of choroidal inflammation using EDI-OCT may potentially be useful in predicting response to therapy.

In patients with unilateral Behcet’s uveitis, subfoveal choroidal thickness in the uveitic eyes was not significantly greater than that of the uninvolved fellow eyes without overt signs of active disease, suggesting that the choroid of the fellow eyes might be equally affected despite the

**TABLE 2.** Correlation Between Changes in Retinal Vascular Leakage and Choroidal Thickness in Behcet’s Uveitis

<table>
<thead>
<tr>
<th>Retinal vascular leakage grade,*</th>
<th>Active Phase</th>
<th>Quiescent Phase</th>
<th>Change</th>
<th>Coefficient of Correlation, P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>mean, (range)</td>
<td>2.11 (1–3)</td>
<td>0.25 (0–1)</td>
<td>1.857 (1–3)†</td>
<td>0.381‡</td>
</tr>
<tr>
<td>Subfoveal choroidal thickness, μm, mean ± SD</td>
<td>398.77 ± 157.39</td>
<td>356.72 ± 141.09</td>
<td>15.3% (0%–61.88%)§</td>
<td></td>
</tr>
</tbody>
</table>

* Retinal vascular leakage:
Grade 0: no sign of any vascular staining or leakage;
Grade 1: staining of vessels with minimal leakage;
Grade 2: more intense leakage with a distinct vascular margin;
Grade 3: even greater leakage with blurring of the large vessel margins.
† Change in leakage (retinal vascular leakage grade in active phase – retinal vascular leakage grade in quiescent phase).
‡ Coefficient of correlation. Spearman coefficient of correlation between change in vascular leakage and % change in choroidal thickness.
§ % Change in choroidal thickness (choroidal thickness in active phase – choroidal thickness in quiescent phase) / choroidal thickness in active phase × 100%.
absence of any apparent inflammation. Given that Behcet’s disease is a systemic disease, one might hypothesize that patients with known Behcet’s disease but without any evidence of ocular involvement could still have subclinical choroidal involvement.

Studies using conventional OCT and A-scan echography have previously shown choroidal thickening in patients with Behcet’s disease, but this methodology has several limitations due to its low resolution. Histopathologic studies are limited in that tissue manipulations during processing, and postmortem changes could significantly alter the in vivo status of the choroid, and cannot be performed in living humans. ICGA is useful in demonstrating choroidal vasculature, but it needs to be performed simultaneously with FA to delineate choroidal lesions separately from the retina, and cannot be readily performed repeatedly due to its invasiveness. In this respect, in vivo EDI-OCT imaging of the choroidal changes in Behcet’s uveitis has several advantages over histopathologic studies and conventional imaging methods in visualizing choroidal structures in a noninvasive and relatively easy way.

Because there was concurrent retinal involvement in Behcet’s uveitis, such as retinal vasculitis, the choroidal thickening observed in the present study may not necessarily represent a primary choroidal inflammation. In our study, most patients showed retinal changes secondary to posterior uveitis or retinal vasculitis. These changes involved varying degrees of vascular leakage concurrent with choroidal thickening. Correlational analysis in this study showed that there was a statistically significant association between EDI-OCT-measured subfoveal choroidal thickness and angiographic changes. Previous studies have suggested infiltration of the choroid with inflammatory cells in association with retinal inflamm-ation but whether the involvement of the choroid is primary or secondary to retinal inflammation could not be investigated in our study.

In our study, Pearson’s correlational analysis revealed no significant relationship between the duration of disease and choroidal thickness (P = 0.222, data not shown). Previously, no significant relationship between disease duration and choroidal hyper/hypofluorescence was identified in simultaneous FA and ICGA studies in Behcet’s uveitis, whereas another study using ICGA showed a significant relationship between the presence of ICG hypofluorescent areas and disease duration.

There are several variables that must be considered in the present study. The retrospective study includes patients from Korean ethnicity only, thus the geographical variability of Behcet’s uveitis needs to be further characterized. Another limitation of the study is that the grading of angiographic finding is subjective and qualitative. In addition, possible correlations with specific findings using ICGA could not be investigated due to the retrospective nature of the study, as not all the patients had the ICGA performed during follow-up. Future studies correlating EDI-OCT findings with ICGA and severity of choroidal inflammation before and after the treatment would be a logical extension of our present studies. Moreover, only the subfoveal choroidal thickness was evaluated, but the subfoveal area might not be the thickest part of the choroid during an acute episode of inflammation, as the choroid is full of vasculature and is a highly anastomosed network of capillaries. Point-to-point measurements provide only limited information about changes in the entire choroid. Future studies investigating choroidal thickness changes at multiple choroidal locations or measurement of choroidal volume.

**Figure 1.** Representative fluorescein angiogram and EDI-OCT of a Behcet’s uveitis patient during active and quiescent phases. There is a severe degree of vascular leakage with blurring of vascular margin (grade 3; A) and marked choroidal thickening is evident during the active phase (subfoveal choroidal thickness = 568 μm; B). In the quiescent phase, there is an improvement of retinal vascular leakage (grade 1; C), along with reduction in subfoveal choroidal thickness (subfoveal choroidal thickness = 439 μm; D). Subfoveal choroidal thickness (solid arrow) is measured from the hyperreflective line of the Bruch’s membrane to the choriocapillary interface (arrowheads).
might provide a more complete description of pathophysiological changes during acute inflammation. In addition, the number of prior recurrences of uveitis was not taken into consideration. Repeated ocular attacks are known to result in irreversible alterations of the retina. Likewise, recurrent attacks could cause irreversible changes in the choroidal interstitium and vasculature, such as intimal thickening and fibrosis, which were not investigated in our study.

TABLE 3. Subfoveal Choroidal Thickness in Both Eyes of Patients with Unilateral Behcet’s Posterior Uveitis Compared With Age-Matched, Sex-Matched, Spherical Equivalent–Matched Normal Eyes

<table>
<thead>
<tr>
<th></th>
<th>Uveitic Eye</th>
<th>Uninvolved Fellow Eye</th>
<th>Normal</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n, eyes</td>
<td>13</td>
<td>13</td>
<td></td>
<td>—</td>
</tr>
<tr>
<td>Age, y</td>
<td>44.31 ± 7.41</td>
<td>44.0 ± 8.22</td>
<td>0.771*</td>
<td></td>
</tr>
<tr>
<td>Sex, Male:Female</td>
<td>8.5</td>
<td>8.5</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Disease duration, y, mean ± SD</td>
<td>6.47 ± 3.35</td>
<td>NA</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Spherical equivalent refractive error, diopters, mean ± SD</td>
<td>−0.82 ± 1.29</td>
<td>−0.17 ± 0.98</td>
<td>−0.69 ± 0.71</td>
<td>0.158*</td>
</tr>
<tr>
<td>Subfoveal choroidal thickness, μm</td>
<td>411.33 ± 144.33</td>
<td>386.92 ± 181.22</td>
<td>236.42 ± 62.1</td>
<td>—</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>391 (232–710)</td>
<td>321 (132–656)</td>
<td>235.5 (129–317)</td>
<td>—</td>
</tr>
<tr>
<td>Median (range)</td>
<td>0.136†</td>
<td>NA</td>
<td>0.001*</td>
<td>—</td>
</tr>
</tbody>
</table>

Dashes in the P value column indicate that there aren’t any P values associated with each parameter listed in the first column (no statistical analysis was performed for those parameters).

* Mann-Whitney U test.
† Wilcoxon signed rank test.

FIGURE 2. Representative fluorescein angiogram and EDI-OCT of a 42-year-old patient with Behcet’s uveitis. During the active phase, fluorescein angiogram shows a moderate degree of retinal vascular leakage (grade 2; A) and subfoveal choroidal thickness is measured at 524 μm (B). In the quiescent phase, retinal vascular leakage shows an improvement (grade 1; C) and choroidal thickening is reduced to 492 μm (D).
there are no published data, the potential drug-induced changes to the choroid by systemic immunosuppressive medications remain to be explored in future studies. Finally, with regard to choroidal vascular involvement, better resolution with deeper penetration would elucidate whether choriocapillaries or larger vessels are mainly involved in the pathogenesis of Behcet’s uveitis.

In conclusion, we demonstrated a significant change of subfoveal choroidal thickness in Behcet’s posterior uveitis as measured by EDI-OCT, which previously could be investigated only by A-scan echography and histological studies. There was a significant correlation between changes in the subfoveal choroidal thickness and changes in the degree of vascular leakage on FA. Subfoveal choroidal thickness measurement by EDI-OCT may therefore be a useful, noninvasive method to evaluate choroidal involvement in Behcet’s uveitis, and could potentially be used to monitor the activity of Behcet’s uveitis in association with angiographic findings. Future studies involving simultaneous choroidal imaging with EDI-OCT and FA/ICGA would help to validate our findings.

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