Subfoveal Choroidal Thickness Changes Following Anti-Vascular Endothelial Growth Factor Therapy in Myopic Choroidal Neovascularization

Seong Joon Ahn,1,2 Kyu Hyung Park,1 and Se Joon Woo1

1Department of Ophthalmology, Seoul National University College of Medicine, Seoul National University Bundang Hospital, Seongnam, South Korea
2Department of Ophthalmology, Armed Forces Capital Hospital, Seongnam, South Korea

Correspondence: Se Joon Woo, Department of Ophthalmology, Seoul National University Bundang Hospital, #300, Gumi-dong, Bundang-gu, Seongnam, Gyeonggi-do 463-707, South Korea; sejoon1@snu.ac.kr.
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Purpose. To investigate subfoveal choroidal thickness (SFCT) changes following intravitreal anti-vascular endothelial growth factor (anti-VEGF) therapy and to identify clinical and choroidal parameters associated with visual outcome in eyes with myopic choroidal neovascularization (CNV).

Methods. In 60 eyes of 54 patients who were treated with anti-VEGF injections for myopic CNV, SFCT was measured using enhanced depth imaging optical coherence tomography at baseline, at 1, 3, and 6 months after initial anti-VEGF therapy, and at the final visit. Subfoveal choroidal thickness was compared between visits in subgroups separated based on anatomic outcome, recurrence, or resolution. Univariate and multivariate regression analyses were performed to identify factors associated with final best-corrected visual acuity (BCVA).

Results. At baseline, the mean SFCT was 47.6 ± 24.7 μm, significantly lower than that of the contralateral eyes (59.8 ± 34.4 μm, P = 0.022). The thickness significantly decreased to 45.2 ± 24.0 μm (P = 0.027) 1 month after the anti-VEGF therapy. In the recurrent cases, the SFCT significantly increased from 46.1 ± 25.5 μm at month 1 to 52.4 ± 25.8 μm at the time of recurrence (P = 0.020); however, no significant change in the SFCT was noted in the nonrecurrent cases. In the regression analyses, the baseline BCVA (P < 0.001) and central macular thickness (CMT; P = 0.005) significantly correlated with the final BCVA, whereas SFCT or its change was not significantly associated with final BCVA.

Conclusions. Subfoveal choroidal thickness significantly decreased following anti-VEGF therapy in myopic CNV, but showed a subsequent increase in recurrence. Subfoveal choroidal thickness may reflect disease activity and aid decision making regarding retreatment in myopic CNV for recurrent cases.

Keywords: antivascular endothelial growth factor, choroidal neovascularization, myopia, optical coherence tomography

Pathologic myopia is prevalent in Asian countries and is characterized by progressive anteroposterior elongation of the eyeball.1–3 In pathologic myopia, various degenerative changes, including chorioretinal atrophy, lacquer crack, staphyloma formation, and diminished choroidal vasculature with altered blood flow, occur in conjunction with the axial elongation.1–3 Choroidal neovascularization (CNV) can also develop in association with pathologic myopia, accounting for 62% of all CNV cases in patients less than 50 years of age,4 and causes severe vision loss in affected eyes.1–6 Owing to myopic CNV, unfavorable visual outcome can occur in untreated eyes and have a significant impact on a patient’s quality of life. Various treatment modalities, including photodynamic therapy (PDT) with verteporfin7–9 and anti-vascular endothelial growth factor (anti-VEGF) agents, have been used and proven safe and effective.10–13

Spectral-domain optical coherence tomography (SD-OCT) can detect morphologic changes in the retina and enable in-depth quantitative analyses in a variety of retinal and choroidal diseases including myopic CNV.14–16 In particular, enhanced depth imaging OCT (EDI-OCT) provides more accurate choroidal assessment by generating higher-resolution choroidal images.14,17 Using EDI-OCT, Ikuno et al.18 identified several morphologic abnormalities of the choroid and suggested that some were risk factors for myopic CNV. Our previous report showed an association between baseline choroidal morphology and treatment outcome following anti-VEGF therapy in eyes with myopic CNV.14

Choroidal changes following anti-VEGF therapy have been reported in other types of CNV, such as polypoidal choroidal vasculopathy (PCV)19 and idiopathic CNV.20 Patients with these types of CNV show significant decreases in subfoveal choroidal thickness (SFCT) following the therapy.15,19,20 In myopic CNV, few studies have described choroidal thickness changes after anti-VEGF therapy.21–23 Ellabban et al.21 reported no significant changes in choroidal thickness following anti-VEGF therapy in patients with myopic CNV. However, Sayanagi et al.22 reported transient choroidal thinning following the therapy. Owing to the limited sample sizes and short-term follow-up periods in these studies, the choroidal changes that
patients were included in our analyses. Posterior border of the choroid (\( n = 3 \)); (2) previous surgery (except cataract extraction), intravitreal injection, or PDT \( (n = 3) \); (3) age \( \geq 75 \) years (for the possibility of age-related CNV etiology) \( (n = 2) \); and (4) poor image quality or poor demarcation of the posterior border of the choroid \( (n = 3) \). Finally, 60 eyes of 54 patients were included in our analyses.

Examinations

Before anti-VEGF therapy, all patients received complete ocular examinations, including Snellen best-corrected visual acuity (BCVA) assessment, slit-lamp biomicroscopy, intraocular pressure (IOP) and axial length measurement, color fundus photography, fluorescein angiography (FA), indocyanine green angiography (ICGA), and SD-OCT (Spectralis OCT; Heidelberg Engineering, Inc., Heidelberg, Germany). Fluorescein angiography and ICGA were obtained using the Heidelberg Retina Angiograph system (Heidelberg Engineering, Inc.) with a confocal scanning laser ophthalmoscope. The size of the CNV before treatment was measured on the FA/ICGA images with embedded software programs. Axial length was measured in all patients using the IOL Master 500 (Carl Zeiss Meditec, Inc., Jena, Germany). After treatment, fundus photography, BCVA assessment, and OCT examination were performed for each patient at monthly intervals up to 3 months after initial treatment and at 3-month intervals thereafter. It was recommended that patients come to the clinic earlier in cases of visual loss with or without metamorphopsia. Additional FA, ICGA, and SD-OCT were performed whenever physicians suspected recurrence of myopic CNV or in cases of visual loss or recurrent metamorphopsia.

Full-thickness choroidal images were obtained using EDI-OCT with eye-tracking and image-averaging systems as described by Spaide et al.\textsuperscript{17} Choroidal thickness was measured manually with calipers as the distance from the outer border of the retinal pigment epithelium to the inner surface of the sclera, as demonstrated in Figure 1, on the horizontal OCT line passing through the fovea. Subfoveal choroidal thickness was measured at the point of the thinnest inner retinal layers that both investigators (SJA and SJW) agreed on as a foveal point before actual measurement. During the measurement, magnified OCT images (225\%) were used to determine choroidal borders and minimize potential errors caused by involuntary movement during manual measurement. Using the same method, the choroidal thicknesses were measured 1 mm from the fovea at the temporal, nasal, superior, and inferior points at baseline and at subsequent visits for supplemental analyses. Central macular thickness (CMT) was measured using a circular map analysis protocol, which measures the distance between the first signal from the vitreoretinal interface and the outer border of the retinal pigment epithelium. An average thickness is then calculated in a 1-mm-diameter circle centered on the fovea. Segmentation errors, if present, were corrected.
TABLE 1. Demographic and Clinical Characteristics of Included Patients

<table>
<thead>
<tr>
<th>Factors</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of eyes</td>
<td>60</td>
</tr>
<tr>
<td>Age, y</td>
<td>58.7 ± 10.1; range, 37–74</td>
</tr>
<tr>
<td>Sex, female (%)</td>
<td>44 (81.5)</td>
</tr>
<tr>
<td>Follow-up period, mo</td>
<td>21.3 ± 12.7; range, 6–60</td>
</tr>
<tr>
<td>Spherical refractive error, D</td>
<td>−12.3 ± 5.0; range, −24.5 to −6.0</td>
</tr>
<tr>
<td>Axial length, mm</td>
<td>29.7 ± 1.6; range, 26.9–33.0</td>
</tr>
<tr>
<td>Lens status, phakia:pseudophakia* (%)</td>
<td>38:22 (63.3:36.7)</td>
</tr>
<tr>
<td>Best-corrected visual acuity at baseline, logMAR</td>
<td>0.83 ± 0.57; range, 20/2000–20/25</td>
</tr>
<tr>
<td>Central macular thickness, µm</td>
<td>353.1 ± 85.0; range, 190–565</td>
</tr>
<tr>
<td>Location of CNV, subfoveal:juxtafoveal (%)</td>
<td>47:13 (78.3:21.7)</td>
</tr>
<tr>
<td>CNV area, mm²</td>
<td>0.82 ± 1.12; range, 0.23–6.5</td>
</tr>
<tr>
<td>Lacquer crack (%)</td>
<td>50 (83.3)</td>
</tr>
<tr>
<td>Myopic degeneration, 1:2:3:4:5 (%)</td>
<td>1:10:29:19:1 (1.7:16.7:48.3:31.7:1.7)</td>
</tr>
<tr>
<td>Posterior staphyloma (%)</td>
<td>57 (95.0)</td>
</tr>
<tr>
<td>Dome-shaped macula (%)</td>
<td>8 (13.3)</td>
</tr>
<tr>
<td>Materials used for injection, bevacizumab:ranibizumab (%)</td>
<td>46:14 (76.7:23.3)</td>
</tr>
<tr>
<td>Number of injections during follow-up periods</td>
<td>2.1 ± 1.5; range, 1–8</td>
</tr>
</tbody>
</table>

* There was no patient in whom cataract surgery was performed during follow-up period.

by manual segmentation before the CMT measurement. The OCT interpretations and measurements were performed by two independent and experienced investigators who were masked to the patients’ clinical information, including information on the disease activity, clinical characteristics, and therapy details. The average of the two measurements was calculated and used for our analyses.

Fundus photographs, FA, and ICGA were used to evaluate the location of CNV and presence of lacquer cracks and to grade myopic degeneration (scale: M0–M5) according to the methods described by Avila et al. Resolution of CNV was evaluated 1 month after treatment and defined as absence of intra-/subretinal fluid on OCT images and no fluorescein leakage. Recurrence of CNV was defined as the recurrence of intra-/subretinal fluid and fluorescein leakage.

**Treatment**

Patients were treated with a single intravitreal injection of 1.25 mg bevacizumab (Avastin; Roche) or 0.5 mg ranibizumab (Lucentis; Novartis) at baseline after topical anesthesia. All injections were given under sterile conditions; after povidone-iodine solution was used to clean the eyelids, a lid speculum was inserted, and the conjunctiva was irrigated with 5% povidone-iodine. The anti-VEGF agent, either bevacizumab or ranibizumab was injected into the vitreous cavity using a 30-gauge needle, at a position 3.5 mm posterior to the corneal limbus in phakic eyes and 3.0 mm posterior in pseudophakic eyes. Retreatment was performed with the drug that was used in the initial injection on an as-needed basis (if CNV recurred or did not resolve after the last injection).

**Statistical Analyses**

Descriptive statistics were obtained for data pertaining to demographics, axial length, CNV location, BCVA at baseline, and presence/absence of posterior staphyloma, lacquer crack, and a dome-shaped macula. The intraclass correlation coefficient (ICC) and Bland-Altman plots were used to examine reproducibility, for example, agreement between the two measurements for choroidal thickness.

Depending on normality of the data, which was determined by results of the Shapiro-Wilk test, a Student’s t-test or Mann-Whitney U test was performed for comparing choroidal thicknesses between independent groups. A paired t-test or Wilcoxon signed-rank test was performed for comparing choroidal thicknesses between paired groups. Baseline SFCT was compared between the eye with myopic CNV and the contralateral eye using the Student’s t-test. Using the paired t-test, choroidal thickness was compared between baseline and 1 month after anti-VEGF therapy. In eyes showing CNV recurrence during the follow-up period, thickness at 1 month after anti-VEGF therapy was also compared with that at the time of CNV recurrence using the Wilcoxon signed rank test.

For statistical analyses for visual outcome, Snellen BCVA results were converted to a logarithm of the minimum angle of resolution (logMAR) value. Visual improvement at follow-up visits was calculated as logMAR (baseline BCVA) – logMAR (follow-up BCVA). Linear regression analyses were used to examine correlations between baseline SFCT or its change at 1 month and final BCVA or visual improvement. Multivariate stepwise linear regression analysis was used to identify the factors that associated significantly with final BCVA. Statistical analysis was performed using SPSS 18.0 for Windows (SPSS, Inc., Chicago, IL, USA), and P values less than 0.05 were considered statistically significant. Continuous data are presented as means ± standard deviation.

**RESULTS**

Demographic and Clinical Characteristics

Baseline demographic and clinical characteristics are presented in Table 1. Mean patient age was 58.7 ± 10.1 years (range, 37–74 years). Forty-four patients (81.5%) were female. Mean spherical refractive error was −12.3 ± 5.0 D (range, −24.5 to −6.0 D), and mean axial length was 29.7 ± 1.6 mm (range, 26.9–33.0 mm). Subfoveal and juxtafoveal CNVs were present in 47 (78.3%) and 15 (21.7%) eyes, respectively. Mean follow-up period was 21.3 ± 12.7 months. The mean number of anti-VEGF injections during the follow-up period was 2.1 ± 1.5 (range, 1–8). The time to recurrence ranged from 2 to 24 months with a mean of 5.9 ± 6.7 months and a median of 3.5 months. Baseline BCVA was 0.83 ± 0.57 logMAR on average, ranging from 20/2000 to 20/25. Lacquer cracks were present in 50 of 60 (83.3%) eyes with myopic CNV.

On average, OCT images were acquired at 12:42 PM, 12:28 PM, 11:42 AM, and 11:56 AM at the baseline and 1-, 3-, and 6-month visits, respectively. In the eyes that showed recurrence,
the OCT images were obtained at 12:35 PM on average. The difference in the OCT image acquisition time between baseline and the 1-month visit was 2 hours and 9 minutes on average, and it ranged from 2 minutes to 5 hours and 5 minutes. The difference between the 1-month visit and the time of recurrence in the eyes with recurrent CNV was 2 hours and 2 minutes, and it ranged from 5 minutes to 5 hours and 1 minute. The maximum difference in the OCT acquisition time between baseline and the follow-up visits in each patient ranged from 32 minutes to 5 hours and 52 minutes, with a mean of 2 hours and 52 minutes.

**Choroidal Thicknesses in Eyes With Myopic CNV Before and 1 Month After Anti-VEGF Therapy**

Figure 1 shows the box plots of baseline SFCT in eyes with myopic CNV and contralateral eyes. There was good interobserver agreement on baseline and follow-up choroidal thickness measurements, with ICC ranging from 0.968 at 1-month measurement (95% confidence interval [CI], 0.943–0.982) to 0.983 at baseline measurement (95% CI, 0.971–0.991). The Bland-Altman plot showed that 3 of 60 points were located outside the 95% limits of agreement (Supplementary Fig. S1). Compared to the contralateral eye (59.8 ± 34.4 μm), eyes with myopic CNV showed significantly thinner baseline subfoveal choroid (P = 0.022 by Student’s t-test). However, there was no significant difference in axial length between the eyes (29.7 ± 1.6 in eyes with myopic CNV versus 29.5 ± 2.5 mm in the contralateral eyes, P = 0.763 by Student’s t-test).

Figure 2 shows OCT images in subgroups separated on the basis of anatomic outcome, resolution, and recurrence. One month after anti-VEGF injection, mean SFCT decreased from 47.6 ± 24.7 to 45.2 ± 24.0 μm (5.0% decrease compared to baseline), which was statistically significant (P = 0.027 by paired t-test), as shown in Supplementary Figure S2. In the 31 fellow eyes in which the OCT images were obtained before and 1 month after the anti-VEGF therapy, however, there was no significant difference in the SFCT before and after the therapy (51.0 ± 26.0 to 50.8 ± 26.2 μm, P = 0.641). In 23 (38.3%) eyes without CNV resolution 1 month after anti-VEGF therapy, SFCT did not differ significantly between before and after anti-VEGF injections (from 47.3 ± 26.4 at baseline to 46.7 ± 26.1 μm at 1 month after treatment, P = 0.460 by Wilcoxon signed rank test). However, in 37 (61.7%) eyes showing CNV resolution 1 month after treatment, a significant difference in SFCT between baseline and the 1-month visit (47.9 ± 25.9 to 44.2 ± 22.9 μm, P = 0.005 by Wilcoxon signed rank test) was observed (Fig. 3).

Twenty-seven eyes with myopic CNV (45%) experienced a mean of 1.48 CNV recurrences. In these eyes, SFCT increased from 46.1 ± 25.5 μm at 1 month after anti-VEGF therapy to 52.4 ± 25.8 μm (14% increase compared to 1-month thickness) at the time of recurrence (Fig. 4), which was statistically significant (P = 0.020 by Wilcoxon signed rank test). In 33 eyes without CNV recurrence, SFCT showed no significant change between 1 month (44.7 ± 22.4 μm) and 3 months (43.1 ± 20.5, P = 0.557) and also between 1 month and 6 months (46.0 ± 22.2, P = 0.343 by Wilcoxon signed rank test) after anti-VEGF treatment.

**Visual Improvement and Its Association With Choroidal Thickness Change**

Anti-VEGF therapy resulted in significant visual improvement from 0.85 ± 0.57 logMAR at baseline to 0.65 ± 0.55 logMAR at the final visit (P < 0.001, paired t-test) in eyes with myopic CNV. Final visual improvement (final BCVA better than baseline) was obtained in 37 (61.7%) patients, whereas 9 (15%) and 14 (23.3%) patients had the same or worse VA at the final visit, respectively. There was no significant
difference in baseline SFCT (48.2 ± 25.7 in eyes with final visual improvement versus 47.0 ± 21.6 μm in those without improvement) or 1-month SFCT change (difference of SFCT between baseline and month 1, 4.6 ± 7.0 vs. 1.2 ± 7.6 μm) between patients with and without final visual improvement (P = 0.965 and 0.266 by Mann-Whitney U test, respectively).

Table 2 presents the association between clinical/choroidal parameters and final BCVA and between the parameters and visual improvement. In univariate regression analyses, final BCVA significantly correlated with baseline BCVA (r = 0.713, P < 0.001) and CMT (r = 0.378, P = 0.003). Choroidal thickness parameters, such as baseline and final SFCT and 1-month SFCT change, showed no significant association with final BCVA or visual improvement (all P > 0.05). In multivariate stepwise regression analyses, a significant association was noted between baseline BCVA and final BCVA (regression coefficient [B] = 0.741, P < 0.001) and between baseline BCVA and visual improvement (B = -0.259, P = 0.042).

**DISCUSSION**

The present study showed that SFCT in eyes with myopic CNV decreased 1 month after anti-VEGF therapy, but increased at the time of recurrence. Our results suggest that choroidal thickness changes may be associated with the pathogenesis and disease activity of myopic CNV.

Maruko and associates reported that SFCT in PCV at baseline was thicker than in normal eyes. The authors suggest that choroidal thickening may be due to choroidal hyperpermeability, which can be noted in central serous chorioretinopathy. Based on their results, they hypothesize that the pathogenesis of PCV is related to choroidal hyperpermeability. In eyes with exudative age-related macular degeneration (ARMD), previous studies reported foveal choroidal thinning. Similar to CNV in exudative ARMD, our results showed that SFCT was thinner in eyes with myopic CNV than contralateral eyes, which is compatible with the finding reported by Ikuno et al. Although it remains unclear why choroidal thinning is associated with the development of myopic CNV, we hypothesize that the pathogenesis of CNV in myopic eyes might be different from that of PCV but similar to that of CNV in exudative ARMD.

However, this study also demonstrated that the SFCT in patients with myopic CNV decreased 1 month after anti-VEGF therapy. Furthermore, it remained thin for up to 6 months after treatment in nonrecurrent cases, whereas the thickness significantly increased at the time of CNV recurrence in recurrent cases. A similar course of choroidal thickness changes has been reported in cases with PCV following PDT and anti-VEGF injection and in those with idiopathic...
CNV and exudative ARMD following intravitreal anti-VEGF therapy. Although the pathogenic mechanisms of CNV formation may differ between some types of CNV and myopic CNV, the CNV that developed may have common properties, as these types of CNV show thinned subfoveal choroids following anti-VEGF therapy and thicker choroids at the time of recurrence. Regressed CNV or reduced choroidal permeability that is associated with reduced VEGF concentrations following anti-VEGF therapy may be plausible explanations for the decreased choroidal thickness. However, increased permeability and subsequent choroidal edema at the time of CNV recurrence may increase choroidal thickness. Clinically, changes in choroidal thickness might be used to guide decisions regarding treatment, especially by identifying cases with recurrent CNV. When treated eyes with myopic CNV show noticeable increases in the SFCT with vision loss, a recurrence of myopic CNV can be suspected. Our results suggested the possibility of choroidal evaluations with EDI-OCT to identify CNV recurrence.

Although eyes with idiopathic CNV showed a significant correlation between visual improvement and 1-month SFCT change, those with myopic CNV showed no association between visual outcome and choroidal parameters. Although CNV activity may decrease following anti-VEGF therapy, the cause of vision loss in eyes with myopic CNV may be associated with other pathologic conditions, such as myopic chorioretinal atrophy, subretinal fibrosis, and cataract, which may result in limited visual improvement following anti-VEGF therapy. Furthermore, RPE atrophy can progress in eyes with myopic CNV, and that, compared to other types of CNV, this effect should be carefully considered for visual prognosis following anti-VEGF therapy.

Several limitations require careful consideration regarding the interpretation of our results. First, the retrospective design results in intrinsic drawbacks, namely, selection bias. Additionally, a relatively small number of patients and variable follow-up periods in our study limit our ability to draw more definite conclusions on final visual outcome after anti-VEGF therapy and its association with SFCT change in eyes with myopic CNV. Although there were no significant differences in baseline characteristics and visual or anatomic outcomes between eyes treated with bevacizumab and those treated with ranibizumab in our study (Supplementary Table S1) and in previous studies, the use of two different drugs may additionally introduce bias.

Additionally, the 2.4-μm difference in the SFCT before and after anti-VEGF therapy might result from measurement error or aging. Furthermore, this small difference may not be sufficient to have a clinical impact or implication. However, the analyses of the four parafoveal choroidal thicknesses (Supplementary Fig. S3) showed a similar trend of choroidal thinning. Therefore, we suggest that the SFCT decrease may be explained by true choroidal thinning rather than by measurement error. Margolis and Spaide reported a 1.56-μm decrease in choroidal thickness per year as a result of choroidal aging in nonmyopic eyes, and this suggests that choroidal aging may explain the choroidal change following anti-VEGF therapy. However, the eyes with very thin choroids in our study may be less likely to show changes of up to 2.4 μm in the SFCT for the 1-month period owing to choroidal aging alone.

Most importantly, diurnal variation may have affected our results, as it is well known that choroidal thickness shows diurnal variation. Tan et al. showed that the highest mean choroidal thickness was obtained at 9:00 AM and that the mean choroid thickness then decreased progressively over subsequent time points to 5:00 PM, which has been confirmed in other studies. It was impossible to control the diurnal variation in every patient of our study because the patients visited our clinic at slightly different times during the day. Despite the limitation of diurnal variation, we evaluated the mean of the visits at which OCT images were acquired to assess whether there was an overall effect of diurnal variation on mean choroidal thickness at baseline and each posttreatment visit. The thickness changes due to diurnal variation between 1 month (mean: 12:32 PM) and the time of recurrence (mean: 12:35 PM) in eyes with recurrence is expected to be insignificant, as the difference in the mean time between the visits was small. Furthermore, as choroidal thickness decreased progressively over the subsequent time points, the thickness at 1 month (mean time of day when OCT was performed: 12:28 PM) was expected to be greater than at baseline (mean: 12:42 PM) due to diurnal variation. However, this was not observed in our cases. Therefore, choroidal thickness changes 1 month after anti-VEGF therapy may be explained as a posttreatment change rather than diurnal variation.

We did not exclude patients with diabetes mellitus or hypertension in our study if they did not show any retinopathy. However, microvascular changes that result from hypertension or diabetes mellitus cannot be excluded, and these changes may have affected the baseline choroidal characteristics in our patients. For a more careful interpretation of our results, the choroidal response to anti-VEGF in patients with hypertension or diabetes mellitus should be investigated in future studies.

In conclusion, our study showed that SFCT decreased after anti-VEGF therapy and subsequently increased at the time of recurrence in eyes with myopic CNV. Choroidal thickness change following anti-VEGF therapy may reflect disease activity in myopic CNV. Thus, it may guide decisions related to retreatment for recurrent myopic CNV cases. Further prospective studies with a larger sample size are necessary to confirm our findings.
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References