의학석사 학위논문

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망막정맥폐쇄 환자에서 맥락막 두께 분석 연구

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지도교수 현 준 영

이 논문을 의학석사 학위논문으로 제출함

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Analysis of Choroidal Thickness in Retinal Vein Occlusion

by

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ABSTRACT

Analysis of Choroidal Thickness in Retinal Vein Occlusion

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Purpose: To measure choroidal thickness in eyes with retinal vein occlusions (RVO) and to compare choroidal thickness before and after the injection of a sustained-release dexamethasone implant.

Method: A retrospective study was conducted on 47 patients diagnosed with unilateral macular edema associated with RVO. All patients were treated using an intravitreal sustained-release dexamethasone implant. Subfoveal choroidal thickness was measured using optical coherence tomography (OCT) before and after dexamethasone implant injection. Each patient was followed up for 5 months after the injection. Subfoveal choroidal thickness of the eye with RVO was compared with that of the contralateral eye, which was normal in all cases. Choroidal thickness was measured at each follow-up examination.

Results: Among 47 patients, 5 were excluded due to missing measurements; 42 patients (89.4%) were finally included. Mean age was 57 ± 13 years. The study population included 25 women and 17 men. Subfoveal choroidal thickness in the eyes with RVO was 254 ± 70 μm, which was higher than that in the contralateral normal eyes (217 ± 55 μm, $p < 0.001$). Choroidal thickness decreased significantly at 1 month, 3 months, and 5 months after the injection of a dexamethasone implant (all $p < 0.001$). Subfoveal choroidal thickness and retinal thickness showed a positive correlation (correlation coefficient: +0.388, $p < 0.001$).

Conclusion: Subfoveal choroidal thickness was greater in eyes with RVO. Choroidal thickness decreased persistently for 5 months after the injection of a sustained-release dexamethasone implant.

Keywords: Choroidal thickness, Corticosteroid, Dexamethasone, Retinal vein occlusion

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CONTENTS

LIST OF FIGURES

INTRODUCTION

Retinal vein occlusion (RVO) is the second most common cause of vascular disease in the retina, after diabetic retinopathy.[1, 2] The risk factors for RVO reportedly include hypertension, arteriosclerosis, diabetes, smoking, hyperlipidemia, inflammatory disease, and hypercoagulable states, in particular, elevated antiphospholipid antibody and plasma homocysteine levels.[3] The standard of care for macular edema in RVO has been grid laser photocoagulation.[4, 5] Recently, intravitreal injection of anti-vascular endothelial growth factor (VEGF) and sustained-release dexamethasone implants have shown promising results in the treatment of macular edema after RVO.[6-8]

After eyes with macular edema (ME) caused by branch RVO or central RVO received sustained-release dexamethasone implants, the time to achieve a ≥15-letter improvement in best-corrected visual acuity (BCVA) was lower in comparison to the time in eyes that underwent sham treatment.[8] The percentage of eyes with a ≥15-letter improvement in BCVA was higher compared with that observed after sham treatment at days 30–90.[8] Severe treatment-related adverse effects, including vitreous hemorrhage, endophthalmitis, and retinal detachment, were extremely rare during the 12-month follow-up period. However, some patients showed signs of cataract progression or increased intraocular pressure.[9]

As optical coherence tomography (OCT) has been developed for choroidal imaging in clinical practice,[10] it has been possible to examine the detailed morphology of the posterior segment in various diseases. Subfoveal choroidal

thickness has been reported to increase in central serous chorioretinopathy,[11, 12] Vogt-Koyanagi-Harada disease, and polypoid choroidal vasculopathy.[13, 14] Choroidal thickness is known to decrease in high myopia[15] and at the site of macular holes.[16] The reports on choroidal thickness in age-related macular degeneration have stated conflicting results.[14, 17]

Choroidal thickness was greater in central RVO as compared to that in fellow eyes and decreased after anti-VEGF treatment.[18] Tsuiki et al. suggested that increased VEGF levels, triggered by tissue hypoxia, induce vessel dilation and enhance vascular permeability, thus increasing subfoveal choroidal thickness.[18] Though steroids are used to cure ME in RVO, no previous report has reported a change in choroidal thickness after intravitreal steroid injection. It has also not been shown whether choroidal thickness would increase again after the recurrence of ME in eyes previously treated for RVO.

This study aims to examine the choroidal thickness changes in eyes with RVO and to compare choroidal thickness measured before and after injection of a sustained-release dexamethasone implant.

MATERIALS AND METHODS

Subjects

We reviewed the medical records of consecutive patients who received intravitreal Ozurdex® (Allergan Inc., Irvine, CA, USA) injections for the treatment of ME associated with retinal vein occlusion at Seoul National University Hospital during the period from November 2011 to January 2013. ME was defined as central subfield retinal thickness (central macular thickness, CFT) ≥ 300 μm, using the macular cube 512 × 128 mode on the OCT (Cirrus high-definition (HD) OCT, Model 4000; Carl Zeiss Ophthalmic Instruments, Dublin, CA; 128 lines, 512 A-scans per line, scan area 6 × 6 mm, Software Version 6.0.2.81). In order to use contralateral eyes as normal controls, those with bilateral RVO were excluded. Only eyes followed for more than 6 months after injection were included. The major exclusion criteria were bilateral retinal vein occlusion; diabetic retinopathy; conditions that affect choroidal thickness, specifically high myopia (>6 diopters), uveitis, and previous history of central serous chorioretinopathy or photodynamic therapy; media opacities that prevented assessment of the fovea, specifically cataract, vitreous hemorrhage, and corneal opacity; previous intravitreal injections of triamcinolone acetonide or bevacizumab (Avastin®; Genentech, Inc., South San Francisco) ≤ 3 months before inclusion; previous pars plana vitrectomy; and a history of any other major surgery including cataract extraction and scleral buckle within the past 6 months. The research was conducted in accordance with the guidelines for the

use of human participants in biomedical research as outlined in the Declaration of Helsinki.

Examinations and data collection

A complete ocular examination, including assessments of BCVA on a Snellen chart, tonometry, biomicroscopy, dilated fundus examination, and OCT imaging, was conducted on each patient. After the Ozurdex® injection, BCVA, tonometry, slit-lamp biomicroscopy, a dilated fundus examination, and OCT were repeated at each follow-up visit. Follow-up visits were scheduled at 1 month after injection, and then every 2 months following. If ME recurred, another injection of Ozurdex® or bevacizumab was administered. The patient was followed-up monthly from that point onward.

Choroidal thickness was measured by spectral domain (SD)-OCT using a Cirrus high-definition (HD) OCT. The scan pattern selected was the 1-line raster, which is a 6-mm line consisting of 20 480 A-scans, an imaging speed of 27 000 A-scans per second, for an average of 20 frames (B-scans). The resultant images were viewed and measured using the appropriate software (version 6.0.2.81; Carl Zeiss Ophthalmic Instruments, Dublin, CA). For the better visualization of the choroidal contour, inverted black-and-white image was selected. The choroid was measured from the outer portion of the hyper-reflective line corresponding to the retinal pigment epithelium to the inner surface of the sclera. Measurements of subfoveal choroidal thickness (SFCT) were obtained at the subfoveal region by manual measurement through calipers provided by the instrument's software. These measurements were performed

by a retinal specialist (JMH). The grader was masked to the diagnosis of each patient.

Statistical Analysis

The Wilcoxon signed-rank test was used to compare the measurements. The correlation between choroidal thickness and retinal thickness was evaluated in eyes with ME associated with RVO. A p value < 0.05 was considered to be statistically significant. Statistical analysis was performed using SPSS 19.0 software for Windows (SPSS Inc., Chicago, IL, USA).

RESULTS

Demographic and clinical characteristics

Among 47 eyes followed up for more than 5 months, 5 eyes were excluded because of failure to demarcate the choroid or failure to measure choroidal thickness; 42 eyes (42/47, 89.4%) were finally included in the study. The study group comprised 25 women and 17 men. The mean age was 57 ± 13 years (range, 25–80). Contralateral normal eyes were assessed as normal controls. The BCVA of RVO eyes ranged from 20/800 to 20/32 (median visual acuity, 20/100). The type of RVO was branch retinal vein occlusion in 26 (62%) patients and central retinal vein occlusion in 16 (38%) patients. Among these patients, 21 (50%) had hypertension and 4 (10%) had diabetes mellitus without retinopathy.

Changes in retinal thickness and choroidal thickness after injection of the sustained-release dexamethasone implant

In the affected eye, mean CMT was 501 ± 148 μm (range, 307–796), and mean SFCT was 254 ± 71 μm (range, 102–352). Mean CMT of the contralateral normal eye was 250 ± 21 μm (range, 192–298), and mean SFCT was 217 ± 55 μm (range, 78–303). These results showed that CMT of the eyes with RVO was higher than that of the contralateral normal eyes ($p < 0.001$). The same trend was observed for SFCT ($p < 0.001$).

CMT decreased at 1 month (298 ± 75 μm, $p < 0.001$) and 3 months (306 ± 85 μm, $p < 0.001$) after injection of the sustained-release dexamethasone implant

(Figure 1). CMT measured at 5 months was 379 ± 154 μm, which was lower than that measured before injection ($p = 0.001$) but higher than that measured at 1 month and 3 months ($p = 0.001$, $p = 0.023$, respectively).

SFCT was 254 ± 70 μm, 233 ± 74 μm, 226 ± 64, and 229 ± 69 μm, at baseline, 1 month, 3 months, and 5 months, respectively (Figure 2). SFCT decreased significantly at 1 month, 3 months, and 5 months (all $p < 0.001$). SFCT measurements at 5 months were similar to those obtained at 1 month ($p = 0.427$) and 3 months ($p = 0.571$).

At baseline, CMT of the eyes with branch RVO was 480 ± 144 μm, and CMT of the eyes with central RVO was 535 ± 175 μm. CMT did not differ between eyes with branch RVO and central RVO ($p = 0.242$). CMT measured at 5 months was 369 ± 145 μm and 395 ± 168 μm, in eyes with branch RVO and central RVO, respectively. CMT did not differ between eyes with branch RVO and central RVO at baseline or at 5 months ($p = 0.242$, $p = 0.777$, respectively). SFCT at baseline was 245 ± 73 μm and 269 ± 71 μm in the eyes with branch RVO and central RVO, respectively. SFCT at 5 months was 227 ± 69 μm and 232 ± 67 μm in eyes with branch RVO and central RVO, respectively. There was no difference in SFCT in eyes with branch RVO and central RVO at baseline or at 5 months ($p = 566$ and $p = 872$, respectively).

Correlation of retinal thickness and choroidal thickness

During the 5-month observation period, SFCT ranged from 63–360 μm (mean, 241 ± 71 μm). SFCT and CMT had a weak positive correlation (correlation coefficient: +0.200, $p = 0.014$). The ratio of the SFCT of the affected eye to that

of the contralateral eye was used to standardize the effect of individual difference. This ratio was 0.7 to 1.6 (mean, 1.1 ± 0.2). The SFCT ratio and CMT had an intermediate positive correlation (correlation coefficient: +0.388, $p < 0.001$). Eyes with branch RVO (26 eyes) and eyes with central RVO (16 eyes) did not differ significantly in SFCT, with adjustment for CMT ($p = 0.775$).

Case

A 65-year old woman with a history of hypertension visited our clinic due to decreased visual acuity in her right eye. She claimed that the visual disturbance had started 1 month prior. BCVA in the right eye was 20/100. Fundus examination showed scattered retinal hemorrhages in all quadrants of the fundus, cystoid macular edema, and venous tortuosity. Fluorescein angiography revealed fluorescein pooling within the macular area. OCT demonstrated diffuse macular edema. CMT was 658 μm and SFCT was 219 μm. The patient was diagnosed with central retinal vein occlusion with macular edema and treated with an intravitreal Ozurdex® injection. After 1 month, visual acuity in the right eye had improved to 20/50. CMT was 318 μm and SFCT was 179 μm. After 3 months, visual acuity in the right eye was 20/32; CMT was 272 μm; and SFCT was 160 μm. After 5 months, recurrent macular edema was observed, and visual acuity of the right eye decreased to 20/40. CMT was 512 μm and SFCT was 188 μm (Figure 3).

Figure 1. Central subfield retinal thickness after the injection of a sustained-release dexamethasone implant. Star (*) indicates $p < 0.05$.

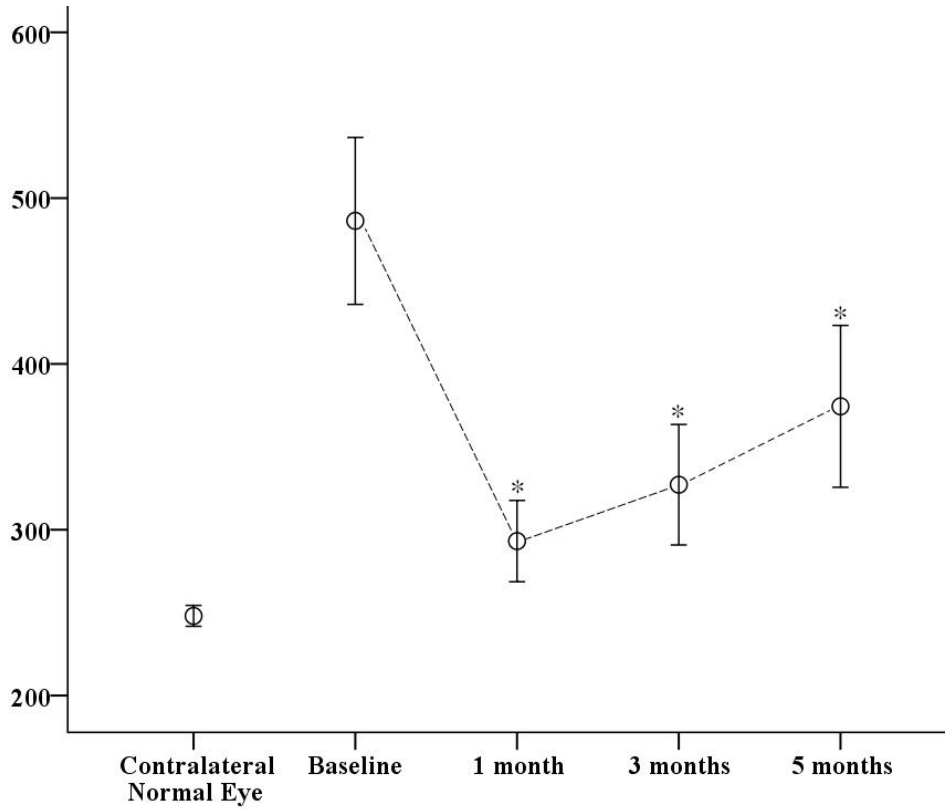


Figure 2. Subfoveal choroidal thickness after the injection of a sustained-release dexamethasone implant. Star (*) indicates $p < 0.05$.

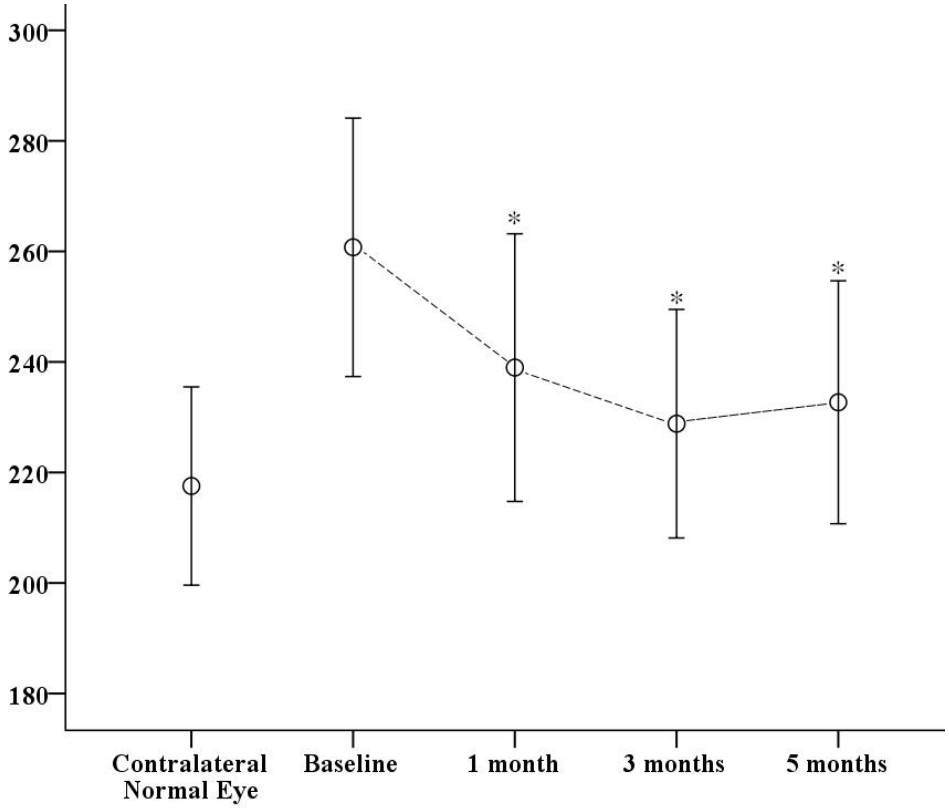
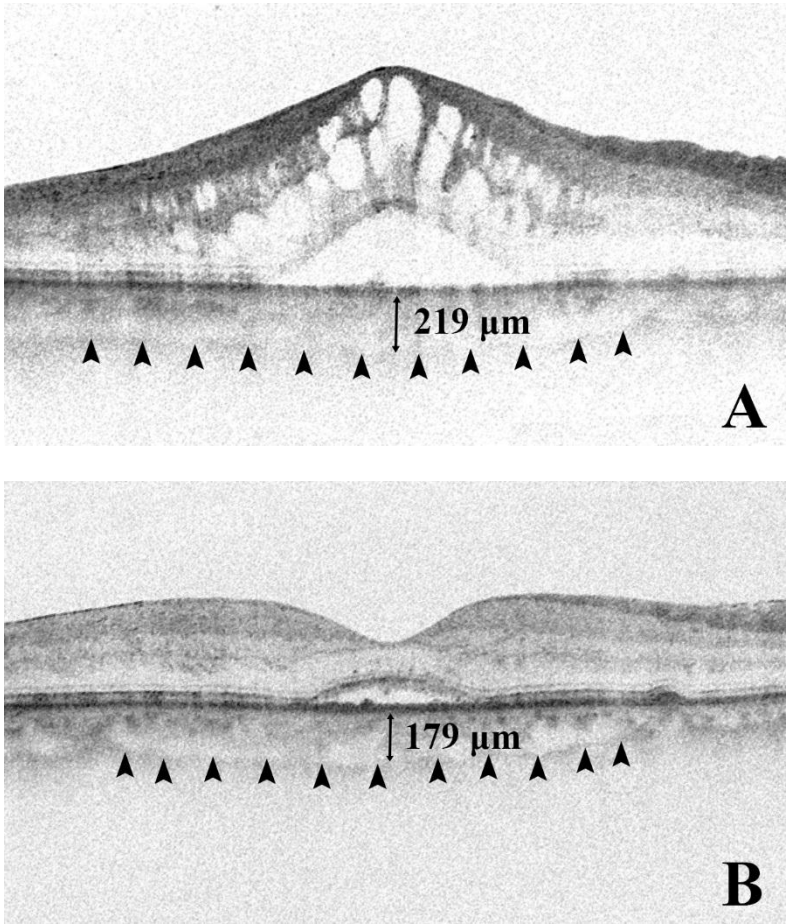
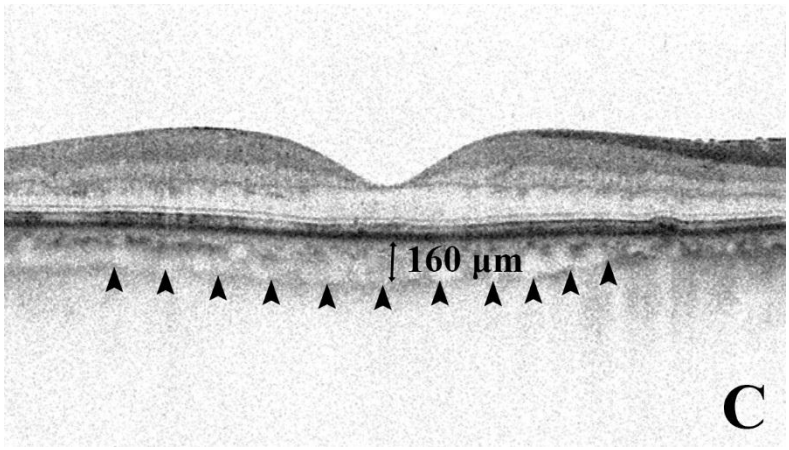
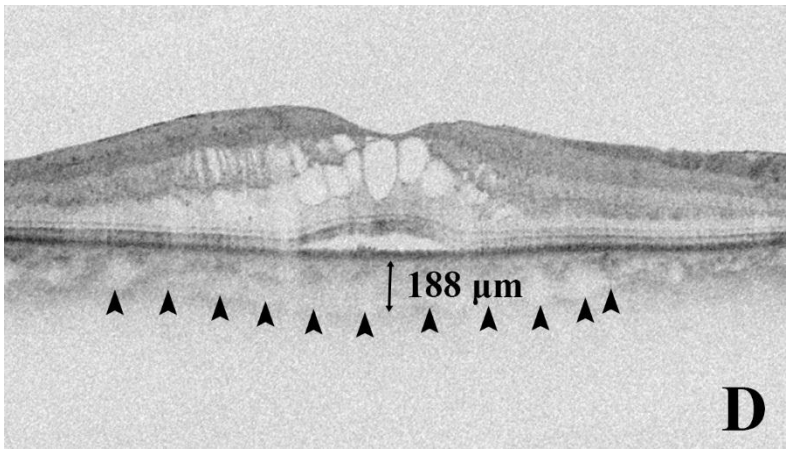


Figure 3. Optical coherence tomography images from a patient that received a sustained-release dexamethasone implant to treat a branch retinal vein occlusion with macular edema. A. Before injection. B. One month after injection. C. Three months after injection. D. Five months after injection. Note that subfoveal choroidal thickness decreased at 1 month and 3 months. Retinal thickness followed the same pattern. Recurrent macular edema was observed at 5 months, at which point subfoveal choroidal thickness had increased slightly but had not returned to baseline levels.




160 µm
C


188 µm
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DISCUSSION

Our measurements of choroidal thickness in RVO eyes with signs of ME revealed subfoveal choroidal thickness to be higher in RVO in comparison with those in normal eyes. After the injection of a sustained-release dexamethasone implant, choroidal thickness decreased, as did central retinal thickness.

Choroidal thickness can be measured using various OCT devices. Spaide et al[10] first reported the use of a Heidelberg Spectralis OCT to measure choroidal thickness with the enhanced-depth imaging (EDI) technique, which measures choroidal thickness by positioning an OCT device close to the eye so as to place the outer choroid closer to the zero-delay line, which results in improved choroidal visualization.[19] Numerous studies on choroidal thickness have been conducted using the Cirrus HD OCT,[20-25] which allows for repeatable measurements.[23] When images of the choroid are captured by Cirrus HD OCT, the inverted images taken in close proximity to the zero-delay have low resolution,[22, 23] resulting in inferior quality compared to the images taken using the Heidelberg Spectralis OCT. It is also possible to average 20 B-scan images in order to measure choroidal thickness.[20, 25] However, using this technique, it is often difficult to identify the boundary between choroid and sclera. Accurate measurements of choroidal thickness were obtained in only 74%[20]–90.7%[23] of cases. In this study, 89.4% (42/47) of choroidal thickness measurements were found to be reproducible.

Mean SFCT, as measured by Tsuiki et al.[18] in CRVO eyes, was 257.1 ± 83.2 μm—greater than that in fellow eyes (222.6 ± 67.8 μm). Mean SFCT decreased

from 266.9 ± 79.0 μm to 227.7 ± 65.1 μm after intravitreal bevacizumab injection. In this study, SFCT in RVO eyes was 254.2 ± 70.4 μm, greater than that in fellow eyes (217 ± 55 μm). After injection of the sustained-release dexamethasone implant, these values decreased to 233.5 ± 73.6 μm and 225.8 ± 64.1 μm at 1 month and 3 months, respectively. A previous study by Tsuiki et al.[18] demonstrated the resolution of choroidal and retinal edema after bevacizumab injection. The results presented here indicate similar results after the injection of a sustained-release dexamethasone implant.

The Beijing Eye Study, a population-based cross-sectional study conducted in northern China, showed that SFCT in eyes with RVO did not differ from that in the normal contralateral eye.[26] This study also showed that SFCT, whether in eyes with branch or central RVO, did not differ from that observed in the normal population, after adjustment for age, gender, axial length, anterior chamber, and lens thickness. In the Beijing Eye Study, there was no instance of marked cystoid macular edema on macular OCT images, and no case of RVO was recent in onset. However, all eyes in our study exhibited macular edema and were treated with sustained-release dexamethasone. Choroidal thickness and retinal thickness showed a positive correlation in eyes with RVO. After adjustment for macular edema, choroidal thickness was not correlated with RVO type (branch or central). The mechanism underlying this increase in choroidal thickness in RVO eyes and the decrease in choroidal thickness after dexamethasone implant injection has not been fully elucidated. The choriocapillaris has fenestrations, which allow for the outflow of large molecules and increase the amount of material leaving the capillaries. Soluble

VEGF isoforms can increase vascular permeability, with these fenestrations disappearing after VEGF withdrawal.[27] In hypoxic RVO eyes, VEGF expression is increased in retinal endothelial cells, pericytes, RPE, Muller cells, ganglion cells, and astrocytes.[28] VEGF induces choroidal vascular hyperpermeability,[18] which in turn increases choroidal thickness.

Choroidal thickening is also mediated by vascular dilation induced by nitric oxide production, which in turn is triggered by VEGF expression.[27] McAllister et al.[28] demonstrated that triamcinolone down-regulates VEGF expression, preventing a decrease in the expression of occludin, a critical component of vascular endothelial tight junctions and essential for the regulation of vascular permeability. This inhibited the expression of GFAP, a protein associated with retinal vascular permeability, in an animal model of BRVO. The mechanism by which dexamethasone reduces choroidal thickness in RVO eyes may involve a similar pathway. The magnitude of the decrease in choroidal thickness observed in our study was similar to that reported previously for the use of anti-VEGF therapeutics.

The correlation between retinal thickness and choroidal thickness was significant but intermediate ($r = 0.388$). Choroidal thickness varied with age,[15] refractive error,[29] axial length,[30] and sex.[31] Although choroidal thickness was adjusted using the ratio of choroidal thickness of the affected eye to choroidal thickness of the contralateral eye, the mechanism of choroidal thickening could differ from that of retinal thickening. The choroid is primarily composed of blood vessels and affected by hemodynamics.[27, 32, 33] Although autoregulatory capacity is limited due to the high partial pressure of oxygen in choroidal tissue,

various studies have suggested that the choroid has some autoregulatory capacity.[34, 35] The numerous factors that affect choroidal thickening include inflammatory cytokines, which may account for the discrepancy between choroidal and retinal thicknesses.

This study has several limitations. First, it was a retrospective study. Second, the sample size was small. Third, choroidal thickness could not be measured in all eyes. The need to exclude 10% of the patients for this reason may have biased the results.

In summary, the choroidal thickness in RVO eyes is greater than that in normal control eyes. However, the injection of a sustained-release dexamethasone implant results in a reduction in choroidal thickness and the resolution of macular edema. Choroidal thickness remained decreased 5 months after injection.

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초 록

목적: 망막정맥폐쇄가 있는 눈에서 맥락막 두께의 변화를 측정하고, 덱사메타손 이식제 주입술이 맥락막의 두께에 미치는 영향을 분석하고자 하였다.

방법: 단안에 발생한 망막정맥폐쇄로, 황반부종의 치료를 위하여 덱사메타손 이식제 주입술 치료를 받은 환자 47 명을 대상으로 후향적으로 연구하였다. 덱사메타손 이식제 주입 전후에 빛간섭단층촬영을 통하여 맥락막 두께를 측정하였으며, 주입술 이후 5 개월간 반복검사를 시행하였다. 망막정맥폐쇄가 있는 눈의 맥락막 두께를 반대편 건측 눈의 맥락막 두께와 비교하였으며, 이후 맥락막두께의 변화를 분석하였다.

결과: 총 47 명의 환자 중 맥락막 두께가 측정되지 않는 5 명의 환자를 제외하고, 42 명(89.4%)의 환자가 최종적으로 포함되었다. 평균 연령은 57 ± 13 년이었다. 25 명의 여자와 17 명의 남자가 포함되었다. 망막정맥폐쇄가 있는 눈의 중심오목하 맥락막 두께는 254 ± 70 μm 로, 건측 눈의 두께인 217 ± 55 μm 보다 두꺼웠다 ($p < 0.001$). 덱사메타손 이식제 주입 후 1 달, 3 달, 5 달 모두에서 맥락막의 두께는

감소한 것으로 측정되었다 (모두 $p < 0.001$). 망막의 두께와 맥락막의 두께는 양의 상관관계를 보였다 (상관계수: +0.388, $p < 0.001$).

결론: 망막정맥폐쇄가 있는 눈에서 중심오목하 맥락막 두께는 정상보다 증가하였다. 중심오목하 맥락막 두께는 덱사메타손 이식제 주입 후에는 5 개월 동안 지속적으로 감소하였다.

주요어: 덱사메타손, 망막정맥폐쇄, 맥락막 두께, 부신피질호르몬

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