Increased Inner Retinal Layer Reflectivity in Eyes With Acute CRVO Correlates With Worse Visual Outcomes at 12 Months

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PURPOSE. To determine if inner retinal layer reflectivity in eyes with acute central retinal vein occlusion (CRVO) correlates with visual acuity at 12 months.

METHODS. Macular optical coherence tomography (OCT) scans were obtained from 22 eyes of 22 patients with acute CRVO. Optical intensity ratios (OIRs), defined as the mean OCT reflectivity of the inner retinal layers normalized to the mean reflectivity of the RPE, were measured from the presenting and 1-month OCT image by both manual measurements of grayscale B-scans and custom algorithmic measurement of raw OCT volume data. OIRs were assessed for association with final visual outcome. Cohort subgroup division for analysis was determined statistically.

RESULTS. Eyes with poorer final visual acuity (≥20/70) at 1 year were more likely to have a higher ganglion cell layer OIR than eyes with better final visual acuity (<20/70) at 1 month (manually: 0.591 to 0.735, P = 0.006, algorithmically: 0.663 to 0.799, P = 0.014). At 1 month, eyes with a poorer final visual acuity demonstrated a higher variance of OIR measurements (algorithmically: 0.087 vs. 0.160, P = 0.002) per scan than eyes with better final visual acuity.

CONCLUSIONS. In acute CRVO, ganglion cell layer changes at 1 month, including increased reflectivity and increased heterogeneity of reflectivity signal as expressed as OIR and OIR variance, were associated with a poorer visual prognosis at 1 year. Technique calibration with larger sample sizes and automated integration into OCT platforms will be necessary to determine if OIR can be a clinically useful prognostic tool.

Keywords: retina, central retinal vein occlusion, optical coherence tomography, image analysis

Central retinal vein occlusion (CRVO) is a visually disabling disorder with a prevalence of 0.1% in individuals older than 40 years, with an estimated 2.5 million adults afflicted worldwide.1,2 Visual outcomes vary significantly depending on presenting visual acuity3 and extent of ischemia seen on fluorescein angiography.4 Additionally, macular edema and conversion from nonischemic to ischemic CRVO may result in a later decline in visual acuity.1,5,6 In the Study of Comparative Treatments for Retinal Vein Occlusion 2 (SCORE2) trial, 34.9% of patients in the aflibercept arm and 38.7% of patients in the bevacizumab arm failed to gain three letter lines despite six monthly injections of anti-VEGF agent.7 This finding highlights our incomplete understanding of this disease process and our inability to accurately prognosticate long-term visual outcomes.

The identification of alternative markers for ischemia has attracted attention over the past several years. Historical approaches quantitated the depth of the relative afferent pupillary defect,8 assessed scotoma size on Goldmann perimeter,9,9 and measured various indices on full-field ERG.9,10 Recent approaches have included measurement of the foveal avascular zone by spectral-domain optical coherence tomography (SD-OCT) angiography,11–13 structural and en face SD-OCT identification of retinal whitening,14,15 and the assessment of foveal pit morphology on SD-OCT images.14 Disruption of the inner retinal layers may manifest as an increased hyperreflectivity signal seen on SD-OCT. This occurs from a mechanical traction, as seen in epiretinal membrane,16 or alternatively, in the setting of ischemia. Acute central retinal artery occlusion demonstrates uniform hyperreflectivity of the ganglion cell layer.17 Paracentral acute middle maculopathy, which is believed to represent focal ischemia at the capillary level, manifests as increased reflectivity at the level of the inner nuclear layer.18 The degree of reflectivity in the inner retina after acute CRVO varies considerably, but has not been evaluated quantitatively to date as a potential visual prognostic marker. This study was designed to quantitatively evaluate inner retinal layer reflectivity in acute CRVO and correlate reflectivity signals at baseline with long-term visual outcomes. To account for the influence of OCT system performance and pre-retinal optical transmission, normalization of inner retinal reflectivity to that of the RPE was performed to create an optical intensity ratio (OIR). We hypothesized that patients with poor vision at 1
year after CRVO would manifest increased inner retinal reflectivity ratios at presentation.

METHODS

The study adhered to the tenets of the Declaration of Helsinki and was approved by the institutional review board of New York University Medical Center. Because of its retrospective nature, informed consent was waived.

Study Subjects

The patient database at a multiprovider referral retinal practice was searched between May 2009 and May 2017 for records with diagnosis codes of CRVO. Patients included in the study were those with acute CRVO who presented within 7 days of decrease in visual acuity, completed follow-up for more than 12 months, and underwent SD-OCT examination at the initial visit and 1 month. Diagnosis of CRVO was established clinically by the identification of retinal hemorrhages, disc edema, and dilated, tortuous veins in all four retinal quadrants. Patients with OCT image quality score <4/10, a history of prior neovascular maculopathy, diabetic retinopathy with macular edema, or ocular trauma were excluded. Clinical data obtained included demographic information; Snellen best-corrected visual acuity (BCVA); time from symptom onset to presentation; number of intravitreal anti-VEGF injections received over 1 year; and the presence of preexisting diabetic retinopathy, cataract, and primary open angle glaucoma. SD-OCT data obtained included the OIRs at baseline and at 1 month (as described below), and the presence of ellipsoid layer loss or attenuation, external limiting membrane (ELM) loss or attenuation, and subretinal fluid at baseline and at 1 month. Follow-up information obtained included Snellen BCVA at 1 year of follow-up and the number and type of anti-VEGF injections, intravitreal corticosteroid injections, laser treatments, incisional glaucoma procedures, and the presence of ellipsoid layer and/or ELM loss or attenuation.

Testing Protocol

All patients included in this study had undergone comprehensive ophthalmic examinations at each visit, including BCVA, applanation tonometry, slit-lamp biomicroscopy, dilated fundus examination, and SD-OCT; 512 × 128 macular cube SD-OCT scans were performed using the Zeiss Cirrus SD-OCT (Carl Zeiss Meditec, Dublin, CA, USA).

Measurement of OIR

Raw OCT data was extracted and imported to ImageJ (http://imagej.nih.gov/ij/; provided in the public domain by the National Institutes of Health, Bethesda, MD, USA) for measurement of the reflectivity in selected retinal layers and calculation of an OIR. Per patient, a single B-scan at the foveal center, superior to the fovea, and inferior to the fovea with at least 50% of the image free of signal abnormality due to blood vessel presence, anterior media shadowing, or intraretinal hemorrhage was selected for OIR measurement. Two authors masked to clinical outcome (NM and FL) manually selected three vertically aligned 10 × 10-pixel areas within the ganglion cell layer (GCL), inner nuclear layer (INL), and RPE to calculate a GCL OIR and an INL OIR by dividing the average intensity of these layers by the mean RPE intensity. The boxes selected were in areas outside of cystoid macular edema and located at least 1.5 mm from the foveal center. This was replicated three times within each B-scan for a total of 9 GCL OIRs and 9 INL OIRs per patient. The mean interobserver difference in OIR measurements was calculated. Sample measurement locations from a single foveal B-scan are presented in Figure 1.

Custom scripting in MATLAB (The MathWorks, Inc., Natick, MA, USA) was then used to algorithmically calculate the entire OIR of a 1 × 1-mm cube of OCT data 1.5 mm temporal, superior, and inferior to the foveal center devoid of macular edema. The original raw OCT cube was de-noised, de-speckled, and smoothed with log and Gaussian filters for peak detection. Peak locations that were not continuous with adjacent A-scan peak locations were excluded to account for layer identification failure. A single OIR from a single A-scan was calculated by dividing mean de-noised inner retina A-scan signal intensity by the mean de-noised RPE A-scan signal intensity. The process was then repeated on all A-scans within the three OCT cubes and approximately 9000 OIRs were averaged to create a final OIR. The SD of values for the scans obtained was calculated to ascertain the variation in reflectivity measurement. Visual quality control was performed by authors NM and MS to ensure correct identification of layers of interest. Visual representation of the algorithm workflow is demonstrated in Figure 2.
To determine a final visual acuity cutoff that most greatly accentuated differences in OIR and OIR variance, the receiver operating characteristic (ROC) curves and area under the curve (AUC) were calculated at 0.1 increments of final visual acuity in logMAR. The stratification with the highest AUC was selected as most predictive. Patients were then arranged into two groups based on that calculated final visual acuity, and an assessment of differential distribution of OIR and OIR variance between the two subgroups was performed via the Mann-Whitney U test. Differential distribution of categorical OCT biomarkers was ascertained by unpaired t-tests. The relationship between qualitative OCT biomarkers and final visual outcome in logMAR was calculated by Mann-Whitney. Finally, linear regression was used to qualify the overall relationship between OIR and final vision (in logMAR). Statistical analyses were performed using GraphPad Prism Version 7 (GraphPad Software, La Jolla, CA, USA).

Of 40 cases reviewed, a total of 22 eyes of 22 (15 male and 7 female) patients were included for further analysis. Eighteen eyes were excluded for poor quality of the SD-OCT scans (16 eyes) and a history of diabetic retinopathy with a diabetic macular edema (2 eyes). The mean age of the entire cohort was 70.6 ± 16.4 years. Mean visual acuity at presentation was 1.12 ± 0.82 logMAR (mean Snellen acuity of 20/260, range 20/40 to hand motions at 2 feet). Nine eyes were pseudophakic. Average time from symptom onset to presentation was 6.5 days. Associated systemic medical findings included hypertension (n = 12), coronary artery disease (n = 11), diabetes (n = 3), and glaucoma (n = 3). Mean overall final visual outcome was 0.74 ± 0.58 (mean Snellen acuity of 20/100, range 20/30 to counting fingers at 2 feet).

**RESULTS**

**Statistical Analysis**

To determine a final visual acuity cutoff that most greatly accentuated differences in OIR and OIR variance, the receiver operating characteristic (ROC) curves and area under the curve (AUC) were calculated at 0.1 increments of final visual acuity in logMAR. The stratification with the highest AUC was selected as most predictive. Patients were then arranged into two groups based on that calculated final visual acuity, and an assessment of differential distribution of OIR and OIR variance between the two subgroups was performed via the Mann-Whitney U test. Differential distribution of categorical OCT biomarkers was ascertained by unpaired t-tests. The relationship between qualitative OCT biomarkers and final visual outcome in logMAR was calculated by Mann-Whitney. Finally, linear regression was used to qualify the overall relationship between OIR and final vision (in logMAR). Statistical analyses were performed using GraphPad Prism Version 7 (GraphPad Software, La Jolla, CA, USA).
Table 1. Patient Demographics and Clinical Characteristics

<table>
<thead>
<tr>
<th>Last Vision</th>
<th>Present (n)</th>
<th>Absent (n)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20/70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. of eyes</td>
<td>10</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Age, y, mean ± SD</td>
<td>75 ± 13.2</td>
<td>70.6 ± 9.1</td>
<td>0.37</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>8</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Presence of subretinal fluid, %</td>
<td>1.1 ± 0.8</td>
<td>1.2 ± 0.7</td>
<td>0.85</td>
</tr>
<tr>
<td>Presence of intraretinal edema, %</td>
<td>75</td>
<td>75</td>
<td>0.04</td>
</tr>
<tr>
<td>Presence of EZ loss/attenuation, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presentation</td>
<td>60</td>
<td>75</td>
<td>0.64</td>
</tr>
<tr>
<td>1 mo</td>
<td>40</td>
<td>83.5</td>
<td>0.07</td>
</tr>
<tr>
<td>1 y</td>
<td>20</td>
<td>66.7</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Presence of ELM loss/attenuation, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presentation</td>
<td>60</td>
<td>75</td>
<td>0.64</td>
</tr>
<tr>
<td>1 mo</td>
<td>40</td>
<td>66.7</td>
<td>0.39</td>
</tr>
<tr>
<td>1 y</td>
<td>20</td>
<td>66.7</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Presence of intraretinal edema, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presentation</td>
<td>100</td>
<td>100</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>1 mo</td>
<td>50</td>
<td>58</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>1 y</td>
<td>20</td>
<td>25</td>
<td>&gt;0.99</td>
</tr>
<tr>
<td>Presence of subretinal fluid, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Presentation</td>
<td>30</td>
<td>75</td>
<td>0.8</td>
</tr>
<tr>
<td>1 mo</td>
<td>41</td>
<td>10</td>
<td>0.16</td>
</tr>
<tr>
<td>1 y</td>
<td>10</td>
<td>25</td>
<td>0.59</td>
</tr>
<tr>
<td>Mean no. anti-VEGF injections at 1 y</td>
<td>6.2 ± 3.2</td>
<td>4.7 ± 2.6</td>
<td>&gt;0.99</td>
</tr>
</tbody>
</table>

Significant P values are bolded.

There were more patients in the poor vision cohort who demonstrated EZ and ELM loss or attenuation at 1 year (P = 0.04, P = 0.04, respectively). The results of demographic and clinical characteristic assessment are presented in Table 1. Vision at 1 year was significantly poorer in eyes with EZ loss/attenuation at 1 month (P = 0.034) and 1 year (P = 0.004) and eyes with ELM loss at 1 year (P = 0.004). Analysis of vision in logMAR stratified by presence or absence of OCT biomarkers is presented in Table 2.

Manual OIR Measurements

At presentation, the mean GCL OIR was 0.709 for the group with final BCVA <20/70 and 0.856 for the group with final BCVA ≥20/70 (P = 0.305). At 1 month, the mean GCL OIR was 0.591 for the better final vision group and 0.735 for the poor final vision group (P = 0.006). The mean INL OIR was similar between the two groups at presentation (0.520 vs. 0.613, P = 0.382) and at 1 month (0.481 vs. 0.573, P = 0.080). The mean interobserver differences between manual measurements across all nine OIRs was 0.037 for the GCL OIR and 0.038 for the INL OIR. Results are displayed graphically in Figure 3.

Custom Scripting

Algorithmically, the mean GCL OIR at presentation was 0.770 for the better final BCVA outcome group and 1.02 for the poorer final BCVA outcome group (P = 0.08). At 1 month, the mean GCL OIR at presentation was 0.663 for the better final BCVA outcome group and 0.799 for the poorer final BCVA outcome group (P = 0.014). The variation, or SD, among all the OIR readings from the presenting OCT was on average 0.164 for the better vision cohort and 0.241 for the poorer vision cohort (P = 0.38). At 1 month, the SD of OIR readings from the better vision group was 0.087 and 0.160 for the poorer vision group (P = 0.002). Results are displayed in Table 3 and Figure 4. Figure 5 highlights the differential variation in OIRs using two sample images. Without subgroup division, initial OIR and OIR variance were positively correlated with a higher 1-year vision in logMAR (slope = 0.90, r^2 = 0.19; slope = 2.19, r^2 = 0.19). One-month OIR and OIR variance were also positively correlated with a higher 1-year vision in logMAR (slope = 2.2, r^2 = 0.25; slope = 4.6, r^2 = 0.28). Graphical results are presented in Supplementary Figure 2. The average difference between OIRs obtained per image between manual and
algorithmic approaches was –0.11. Bland-Altman representations of the results are displayed in Figure 6.

**DISCUSSION**

The current study quantitated inner retinal reflectivity via an OIR in patients with CRVO. The authors developed and used a proof-of-concept custom algorithm to automate the OIR measurement across multiple A-scans within a volume of OCT data. At 1 month after presentation, via both manual and algorithmic segmentation analysis, the reflectivity ratio comparing the GCL with the RPE was greater in eyes with worse final visual acuity than in eyes with better final visual acuity using a statistically derived visual acuity cutoff of Snellen 20/70. Additionally, when evaluating all eyes together, increasing OIR and OIR variance at 1 month were correlated with decreasing visual acuity at 1 year.

The authors sought to evaluate the prognostic significance of acute ischemic insults in CRVO as manifested by retinal hyperreflectivity and retinal layer disorganization as seen on the OCT. Given that reflectivity signals diminish progressively after the acute insult, we selected patients who were within 7 days of symptom onset. Interestingly, at presentation, OIR and OIR variance were not significantly prognostic of visual acuity at 1 year. However, at 1 month after initial visit, both OIR and OIR variance were prognostic of 1-year visual acuity. One explanation for this may be related to the extent of macular edema at presentation versus at 1 month after initial anti-VEGF treatment. At presentation, there may be extensive macular edema and thus the manual calculations and algorithm are capturing hyporeflective cystoid spaces, which would artifi-

**TABLE 3.** Algorithmic OIR Measurement Stratified by Last Vision

<table>
<thead>
<tr>
<th></th>
<th>Presenting OCT</th>
<th>One-Month OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Last VA &lt; 20/70</td>
<td>Last VA ≥ 20/70</td>
</tr>
<tr>
<td>Temporal cube OIR</td>
<td>0.714</td>
<td>0.846</td>
</tr>
<tr>
<td>OIR variance</td>
<td>0.159</td>
<td>0.260</td>
</tr>
<tr>
<td>Superior cube OIR</td>
<td>0.740</td>
<td>1.085</td>
</tr>
<tr>
<td>OIR variance</td>
<td>0.177</td>
<td>0.204</td>
</tr>
<tr>
<td>Inferior cube OIR</td>
<td>0.857</td>
<td>1.065</td>
</tr>
<tr>
<td>OIR variance</td>
<td>0.157</td>
<td>0.282</td>
</tr>
<tr>
<td>Average OIR</td>
<td>0.770</td>
<td>1.020</td>
</tr>
<tr>
<td>OIR variance</td>
<td>0.164</td>
<td>0.241</td>
</tr>
</tbody>
</table>

Significant P values are bolded. VA, visual acuity.

![Figure 3](https://example.com/figure3.png)
FIGURE 4. (A) Mean inner retinal OIR at presentation was 0.770 for the better final BCVA outcome group and 1.020 for the poorer final BCVA outcome group ($P = 0.08$). (B) At 1 month, the mean inner retinal OIR at presentation was 0.663 for the better final BCVA outcome group and 0.799 for the poorer final BCVA outcome group ($P = 0.014$). (C) The variation, or SD, among all the OIR readings from the presenting OCT cube was on average 0.164 for the better vision cohort and 0.241 for the poorer vision cohort ($P = 0.38$). (D) At 1 month, the SD of OIR readings from the better vision group was 0.087 and 0.160 for the poorer vision group ($P = 0.002$).

FIGURE 5. Top row: One-month foveal B-scan images. Bottom row: Histogram representation of all 9000 OIRs obtained from all A-scans within the volume OCT data. Both patient A and patient B presented with 20/400 vision, macular edema, and EZ loss; however, vision at 1 year was 20/40 for patient A and 20/100 for patient B. Patient A’s OIR mean was 0.67 and SD was 0.07 versus 0.73 and 0.15 for patient B.
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**Figure 6.** Bland-Altman plot comparing the average OIR per OCT by two methods plotted against the difference between manual and algorithmic measurements demonstrating a lower difference between the two methods at lower average OIRs, and an overall trend toward higher OIRs measured algorithmically. As well, the difference between manual and algorithmic measurements increased as average OIR increased.

Browning et al.\textsuperscript{14} recently used a qualitative analysis and ischemia grading scale to evaluate inner retinal reflectivity in CRVO. Although they demonstrated intergrader reproducibility, they did not demonstrate a correlation with functional outcomes. Differences between their study and the current study include a quantitative approach to the latter, which may highlight differences that may not otherwise be perceptible from a purely qualitative approach. Also, analysis of optical intensity is subject to significant variation based on media opacity and incident light.\textsuperscript{25,26} Finally, nonlinear postprocessing applied by commercial OCT software further prevents intersubject signal comparisons. The current study used a normalization ratio to allow for consistent optical intensity measurements through a ratio (the OIR) and used raw OCT data to bypass postprocessing images. Further advantages to our study include interobserver correlation for the manual measurements and correlation between manual and algorithmic methodologies.

Our study carries several limitations beyond the retrospective nature of the design and small sample size. First, we established binary groups with a cutoff at 20/70 based on our AUC analysis. Although this was designed to establish a biomarker threshold, it was established in a small sample size and may not be the “true” visual acuity differentiator in a larger sample size. Additionally, despite a correlation between increasing OIR and worse visual acuity across all data points, there is a fair amount of “noise” in the data. It remains unclear why one eye may deviate from these trends and have a high OIR but good final visual acuity and vice versa. To use OIR as a clinically relevant imaging biomarker, future studies with larger sample sizes would allow for a more robust statistical calculation of the most appropriate and prognostic cutoff and to potentially better understand the outliers in this cohort.

Second, our normalizing factor was the RPE, as we wanted to normalize to the highest reflectivity structure. Chen et al.\textsuperscript{27} examined the reflectance levels in normal eyes and identified the RPE as the structure with the consistently highest intensity values. However, the usage of the RPE in pathological retinas opens the study to both hyperreflective bias due to cystoid spaces (as described earlier) or hyperreflective biases.

One hyperreflective bias that would be expected to be similar between both groups of eyes in our study would be the inadvertent inclusion of hyperreflective anatomy in the cropped cube (such as superficial blood vessels). On the other hand, a hyperreflective bias due to shadowing of the RPE from an overlying highly reflective inner retinal tissue would possibly be more prevalent in the ischemic cohort. Bland-Altman analysis helps reinforce this notion by noting that although the difference between manual and algorithmic measurements in our study was low, there was an overall trend toward higher OIRs measured algorithmically. Additionally, the difference between manual and algorithmic measurements increased as the average OIR increased. This can perhaps be explained by a bias to manually measure the OIR in a region of the retina that has good signal strength with potentially less RPE shadowing. When the algorithm automatically measures through that same area of highly reflective tissue, the areas of shadowed RPE drive the OIR of the total region higher. As the ischemia increases, shadowing results in an overall increased OIR as measured by algorithmic versus a manual approach. Unlike hyperreflective bias, this phenomenon drives apart differences between lower and higher levels of ischemia. Our cohort included several cases of severe ischemia, perhaps accentuating the differences between the two cohorts. To circumvent this problem, other studies have suggested the use of the saturation reflectivity or “pure white” in a false color scheme of an OCT image as the
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 normalization factor.29 Further studies will be needed to refine the measurement technique of OCT intensity.

Last, we arbitrarily designated our measurement zones as 1.5 mm away from the fovea and did not include the foveal center. This rationale was predicated on the premise that CRVO induces a pan-macular inner retinal ischemia. Thus, the cube sizes and location were chosen to minimize macular edema and inclusion of blood vessels to reduce both the hyporeflective and hyperreflective biases, respectively. Future directions of this study would include the ability to automatically segment out cystoid spaces and hyperreflective anatomy from the OCT cube and calculate OIRs from larger areas to further help understand the significance of OIR in acute ischemia.

In conclusion, quantitative analysis of inner retinal hyperreflectivity performed in this study indicates that increased inner retinal hyperreflectivity and increased variability in inner retinal reflectivity at 1 month may prognosticate poorer vision at 1 year. Technique calibration with larger sample sizes will be necessary to determine if OIR and OIR variation can be clinically useful tools.

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References