Comment. Cyclodialysis clefts occur most commonly after trauma and sometimes after surgery. Intentional surgical cyclodialysis has been used for the treatment of glaucoma.2,6 Gonioscopy has been the classic method for visualizing a cyclodialysis cleft; however, cyclodialysis clefts could be missed in traumatized eyes with hazy media, distorted anatomy, or hypotonia. In these cases, UBM has been useful.2,5

We presented diagnostic images of a cyclodialysis cleft using a new technique, anterior segment OCT. This technique provided high-resolution images of the cyclodialysis cleft, showing its exact location and the extent of the disinsertion of the ciliary body from the scleral spur and an associated ciliary body detachment. These images served to confirm the clinical diagnosis.

Anterior segment OCT is a noninvasive tool that provides accurate and reproducible images of the anterior segment. This technique has good correlation with UBM and allows observation of iridocorneal angle and an associated ciliary body detachment. These images served to confirm the clinical diagnosis.

Figure 3. A cyclodialysis cleft (arrows) confirmed by anterior segment optical coherence tomography. Disinsertion of the ciliary body from the scleral spur and an associated ciliary body detachment were also revealed.

in that eye. This result suggested that the patient had lost his accommodation, probably because of the ciliary body detachment. The IOP in the left eye was 10 mm Hg, and macular folds were less marked. Repeated UBM and anterior segment OCT showed persistence of the cyclodialysis cleft and an even larger ciliary body detachment.

The patient’s vision status was excellent compared with that of other patients with cyclodialysis clefts described in the literature.7 Because visual acuity was good and IOP was normal, continued follow-up was agreed on. However, the patient was informed of the risk of continued enlargement of the cyclodialysis cleft; if this were to happen, different treatment options would be proposed, such as suturing the ciliary body to the sclera, vitrectomy with pars plana cryopecty, different treatment options such as gas tamponade8 or argon laser photoocoagulation.9

60-3 Long-Chain Polyunsaturated Fatty Acid Intake Inversely Associated With 12-Year Progression to Advanced Age-Related Macular Degeneration

Age-related macular degeneration (AMD) is a leading cause of vision loss. Age-Related Eye Disease Study (AREDS) participants reporting the highest intake of ω-3 long-chain polyunsaturated fatty acids (LCPUFAs) were approximately half as likely as their peers reporting the lowest intake of these nutrients to have neovascular (NV) AMD1 at baseline or to progress across a 6-year period from bilateral drusen to central geographic atrophy (CGA).2 The Age-Related Eye Disease Study provides data that represent, to our knowledge, the largest longitudinal sample collected and classified with standardized methods as part of a natural history study on AMD. We now report that our baseline and 6-year findings persisted in 12-year AMD incidence models. These results are consistent with existing data.3,4

Methods. The Age-Related Eye Disease Study was a National Institutes of Health–sponsored and administered multicenter study designed to assess the clinical course, prognosis, and risk factors of AMD (http://www.nei.nih.gov/amd/). We examined the relationship of dietary intake of ω-3 LCPUFAs with progression to advanced AMD in 1837 AREDS participants who had a moderate risk for developing sight-threatening AMD (1211 participants in category 3a and 626 participants in category 4a). Participants in category 3a had bilateral visual acuity of 20/32 or better and bilateral large (≥125-μm) drusen, extensive intermediate drusen, and/or geographic atrophy that did not involve the center of the macula in at least 1 eye. Category 4a participants had visual acuity of 20/32 or bet-
ter and no advanced AMD (geographic atrophy involving the center of the macula or features of NV AMD) in the study eye; the fellow eye had definite lesions of advanced AMD. Category 3b and 4b participants had visual acuity worse than 20/32. In AREDS report No. 1,3 we described outcome ascertainment for CGA and NV AMD from centralized grading of annual stereoscopic fundus color photographs; participants progressing to CGA and/or NV AMD in their study eye(s) were classified with incident advanced AMD. We estimated dietary intake with a validated semiquantitative food frequency questionnaire developed for AREDS.1,2 Nutrient density values at baseline defined the LCPUFA variables.3 We computed odds ratios in repeated-measures logistic regression models incorporating generalized estimating equation methods. This permitted determination of advanced AMD at each visit for each participant. All of the models included terms for baseline age (<65 vs 65-69 and ≥70 years), sex, smoking status at diagnosis (never, past, or current), total energy intake (modeled as a continuous variable), AREDS treatment (placebo vs zinc, antioxidants, and zinc plus antioxidants), and baseline AMD status (AREDS category 3a vs 4a).

Results. Participants reporting the highest baseline consumption of ω-3 LCPUFAs were approximately 30% less likely than their peers reporting the lowest ω-3 LCPUFA consumption to develop advanced AMD by the end of the 12-year follow-up period (Table). Results for CGA and NV AMD were similar; respective multivariate odds ratios were 0.63 (95% confidence interval, 0.49-0.94; P = .029) and 0.029, 0.041, and 0.072, respectively, for participants in the highest quintile.

Comment. ω-3 LCPUFAs and their metabolites have the capacity to act on processes implicated in AMD pathogenesis.3 Although inferences are constrained by the observational nature of our research designs (frequent consumption of ω-3 LCPUFA–rich foods may be a proxy for exposure to unmeasured environmentally or behaviorally based protective factors), biologically credible explanations for relationships between ω-3 LCPUFAs and AMD now exist in studies applying in vivo6 and in vitro7 model systems. The findings from these basic studies strengthen conclusions from extant observational studies (reviewed by SanGiovanni and Chew3 and Chong et al7) regarding the association of ω-3 LCPUFA intake with AMD. Because the concentration of retinal ω-3 LCPUFAs is modifiable by and dependent on dietary composition, these nutrients may represent an easily implemented approach to modifying risk of AMD progression; we are now conducting a 5-year, 4000-person clinical trial to examine this issue of public health significance (http://www.areds2.org).

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Table. Odds Ratios for 12-Year Progression to Advanced Age-Related Macular Degeneration

<table>
<thead>
<tr>
<th>Intake Quintile, %</th>
<th>Progression to Advanced AMD, OR (95% CI)b</th>
<th>P Valueb</th>
</tr>
</thead>
<tbody>
<tr>
<td>ω-3 LCPUFA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EPA Bottom 20</td>
<td>0.69 (0.53-0.90)</td>
<td>.007</td>
</tr>
<tr>
<td>DHA Bottom 20</td>
<td>0.67 (0.51-0.88)</td>
<td>.004</td>
</tr>
<tr>
<td>EPA + DHA Bottom 20</td>
<td>0.65 (0.50-0.85)</td>
<td>.002</td>
</tr>
</tbody>
</table>

Abbreviations: AMD, age-related macular degeneration; CI, confidence interval; DHA, docosahexaenoic acid; EPA, eicosapentaenoic acid; LCPUFA, long-chain polyunsaturated fatty acid; OR, odds ratio.

aAdvanced AMD includes central geographic atrophy and/or choroidal neovascularization. Medians of reported daily intake values for EPA, DHA, and EPA + DHA in percentage of total energy intake are 0.002, 0.007, and 0.009, respectively, for participants in the lowest quintile of the Age-Related Eye Disease Study sample and 0.029, 0.041, and 0.072, respectively, for participants in the highest quintile.

bComparisons are for the lowest quintile vs the highest quintile (group A is the reference category).
Vascular Dilation and Tortuosity in Plus Disease

Plus disease is the major factor in determining whether peripheral retinal ablation for retinopathy of prematurity is needed. Although the clinical definition of plus disease includes both dilation and tortuosity, previous reports have suggested that computer-based programs assessing tortuosity alone may be adequate to diagnose plus disease. The aim of this study was to determine the frequency of isolated dilation sufficient for plus disease (with insufficient tortuosity) and isolated plus-level tortuosity (with insufficient dilation) compared with the frequency of both plus-level dilation and plus-level tortuosity in a series of images.

Methods. In a previously published, institutional review board–approved study designed to compare the accuracy of computer-assisted image analysis using ROPtool with that of individual examiners, 6 pediatric ophthalmologists (S.F.F., Terri L. Young, MD, Laura B. Enyedi, MD; Graham E. Quinn, MD; Michael F. Chiang, MD, MA; and David K. Coats, MD) evaluated RetCam photographs of 190 different eyes from 117 premature infants. Of these photographs, 10 were excluded because of inadequate image quality as determined by one of us (D.K.W.) and 110 were excluded because they were determined to be without either dilation or tortuosity sufficient for plus disease by all of the experts. Therefore, a total of 70 images of 70 different eyes were included. The ophthalmologists independently scored each quadrant of each image by grading dilation and tortuosity separately (8 total grades per eye) as plus, preplus, or normal.

These scores were used to generate eye-level data. An eye-level grade of dilation (or tortuosity) sufficient for plus disease was present if at least 2 of the 4 quadrants in a single eye had dilation (or tortuosity) sufficient for plus disease. These grades were used to determine whether eyes had plus disease (2 quadrants of plus dilation and tortuosity), dilation sufficient but tortuosity insufficient for plus disease, tortuosity sufficient but dilation insufficient for plus disease, or dilation and tortuosity insufficient for plus disease. Thus, the reference standard for plus disease was dilation and tortuosity in the same 2 quadrants, which was the same definition used to define plus disease in both the Early Treatment for Retinopathy of Prematurity and Supplemental Therapeutic Oxygen for Prethreshold Retinopathy of Prematurity clinical trials. The same guideline was used for preplus disease, which we defined as in our previous studies as at least 2 quadrants of preplus dilation and tortuosity worse.

For each ophthalmologist, the specificity of using dilation and tortuosity alone to diagnose plus disease was calculated, with both dilation and tortuosity as the reference standard. Therefore, each individual rater using only dilation or tortuosity was compared against himself or herself using both dilation and tortuosity. In this way, it was determined how often dilation was sufficient but tortuosity was insufficient for plus disease and how often tortuosity was sufficient but dilation was insufficient for plus disease (Figure). Specificity was calculated by dividing the number of eyes classified as not having plus disease as determined by dilation or tortuosity alone by the true number of eyes without plus disease as determined by dilation and tortuosity together. The identical analysis was also performed for the preplus level of disease or worse including the 70 images that were used in the plus analysis and the 110 images without dilation or tortuosity that were excluded from the plus analysis.

Figure. Examples of RetCam photographs used for the study. A, Dilation sufficient but tortuosity insufficient for plus disease (image courtesy of Michael Chiang, MD, MA). B, Tortuosity sufficient but dilation insufficient for plus disease (image courtesy of PHOTO-ROP).