associated with microphthalmos and a cyst contains CSF-like fluid based on positivity for \(\beta_2\)-transferrin, an assay used routinely to detect CSF leak following basal skull fracture. It was a surprise, therefore, that, in case 1, cisternography failed to verify a communication between the subretinal space and intracranial CSF. The most likely source of the fluid is the orbital cysts that are intimately related to the retrobulbar optic nerve in all 3 eyes with retinal detachment. The right eye of case 1 has not yet developed retinal detachment and has a small cyst anteroinferiorly in the orbit. This hypothesis is supported by clinicopathological correlation in which a blind microphthalmic eye-cyst complex was excised. The cyst contained \(\beta_2\)-transferrin–rich fluid that was lined by ependymal cells, similar to those of the choroid plexus, which produce CSF intracranially. In normal eyes, the outer retina, including the retinal pigment epithelium, terminates at the edge of the optic disc. Case 2 confirms that the retina herniates posteriorly through the disc excava
tion where the subretinal space presumably communicates with the cyst cavity. The series of fundus changes in the left eye of case 2 suggests that the continuous presence of subretinal fluid is potentially detrimental because it is associated with a pigmented response from the retinal pigment epithelium. Based on our hypothesis, the net removal of fluid from the subretinal space could be favored by reducing CSF production using oral acetazolamide or by surgically shunting subretinal fluid, for example by a guarded posterior sclerostomy. If an isolated choroidal coloboma.

In summary, we have a new hypothesis explaining retinal detachment in some cases of optic nerve colobomas, which in our view mandates orbital imaging to determine the presence of cysts that could be producing a \(\beta_2\)-transferrin–rich fluid. Vitrectomy may be an inappropriate technique to use. Other techniques could be considered if spontaneous improvement does not occur because the retinal pigment epithelium may be compromised and result in poor visual outcome.

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Longitudinal Study of Retinal Nerve Fiber Layer Thickness and Ganglion Cell Complex in Traumatic Optic Neuropathy

Optical coherence tomography (OCT) has been developed to objectively evaluate structural damage in vivo. The tool can be used to evaluate damage to the circum papillary retinal nerve fiber layer (cpRNFL). Measurements of the cpRNFL play an important role in the diagnosis and management of patients with glaucoma. The recent introduction of spectral-domain OCT has enhanced the scan resolution and provides more reproducibility for image acquisition compared with time-domain OCT, a previous version of OCT. Furthermore, the RTVue (Optovue Inc), one of the spectral-domain OCT instruments, allows us to evaluate inner retinal layer thickness. Reference to the ganglion cell complex (GCC) includes the retinal nerve fiber layer, retinal ganglion cell layer, and inner plexiform layer. Some studies have demonstrated the usefulness of GCC as well as cpRNFL thickness as measured by RTVue in treating glaucomatous eyes.

Traumatic optic neuropathy (TON) causes acute axonal loss with severe vision loss. A few studies have demonstrated axonal loss through the use of time-domain OCT or scanning laser polarimetry after TON. Recently, Cunha et al compared cpRNFL loss and macular thinning in 3 patients with TON. However, to our knowledge, no study has performed a direct comparison between cpRNFL thickness and GCC after TON. We herein statistically evaluated longitudinal changes in cpRNFL thickness and GCC in 4 patients with unilateral TON.

Report of Cases. Subjects were recruited for this observational study from Kobe University Hospital (Kobe, Japan). The study protocol was approved by the institutional review board of Kobe University and ad-
hered to the tenets of the Declaration of Helsinki. Written informed consent was obtained from each subject after explanation of the study.

Four patients with unilateral TON received full ocular examinations repeatedly for 8 months after the trauma. All patients had a history of indirect TON without any intraocular disease and any blowout orbital or optic canal fracture. All patients showed acute and severe visual loss with relative afferent pupillary defects in the affected eyes. The affected eyes did not have other ocular diseases and their intraocular pressure was normal. The contralateral eyes did not display ocular disease or provide abnormal visual field findings. The patient characteristics and visual outcomes are summarized in the Table.

The optic nerve head map protocol was applied to evaluate the cpRNFL. This protocol generates a cpRNFL thickness map based on measurements obtained along a circle 3.45 mm in diameter centered on the optic disc. The GCC protocol was used to determine the GCC and macular thickness. Only high-quality images, as defined by a signal strength index more than 30, were accepted. The first examination was performed at 1 week after injury. The subsequent examinations were performed at 2, 3, 4, 12, and 20 weeks after injury as well as at the final visit (32 to 36 weeks after injury). The reduction occurred 2 to 8 weeks after the injury and had almost completely disappeared at 20 weeks. The cpRNFL measurements exhibited significantly greater reductions than did the GCC and macular thickness. The final relative ratio of cpRNFL thick-

<table>
<thead>
<tr>
<th>Case/Sex/Age, y</th>
<th>Eye</th>
<th>Initial VA</th>
<th>Final VA</th>
<th>Type of Injury</th>
<th>Average cpRNFL Thickness, µm</th>
<th>GCC, µm</th>
<th>Macular Thickness, µm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/36</td>
<td>Right</td>
<td>NLP</td>
<td>NLP</td>
<td>Head trauma after falling</td>
<td>52.25</td>
<td>55.64</td>
<td>227.08</td>
</tr>
<tr>
<td>2/M/27</td>
<td>Right</td>
<td>14/20</td>
<td>20/20</td>
<td>Head trauma after bicycle fall</td>
<td>60.17</td>
<td>62.69</td>
<td>233.95</td>
</tr>
<tr>
<td>3/F/53</td>
<td>Right</td>
<td>NLP</td>
<td>4/400</td>
<td>Stab injury to the orbit</td>
<td>57.31</td>
<td>65.3</td>
<td>231.2</td>
</tr>
<tr>
<td>4/M/50</td>
<td>Left</td>
<td>NLP</td>
<td>NLP</td>
<td>Head trauma by automobile</td>
<td>62.44</td>
<td>74</td>
<td>244.37</td>
</tr>
</tbody>
</table>

Abbreviations: cpRNFL, circumpapillary retinal nerve fiber layer; GCC, ganglion cell complex; NLP, no light perception; VA, visual acuity.

Figure 1. Changes in the optic nerve head map (A) and ganglion cell complex significance (B) of the right eye in case 1. Average circumpapillary retinal nerve fiber layer thickness and ganglion cell complex thickness in each session are also shown. IN indicates inferior nasal; IT, inferior temporal; NL, nasal lower; NT, nasal temporal; NU, nasal upper; SN, superior nasal; ST, superior temporal; T, temporal; TL, temporal lateral; and TU, temporal upper.
circumpapillary retinal nerve fiber layer (cpRNFL) thickness, average ganglion cell complex (GCC), and macular thickness in 4 eyes after traumatic optic neuropathy. *Paired t test, P<.05 compared with the contralateral eyes.

Comment. Given that it is impossible to longitudinally perform histological examinations in humans, in vivo imaging is a valuable technique. Previous studies have explored reductions in cpRNFL thickness after TON. Three studies using scanning laser polarimetry reported that cpRNFL thickness did not decrease within 2 weeks after injury.1-3 On the contrary, 2 studies using time-domain OCT demonstrated that the cpRNFL returns to normal thickness within 1 week. Cunha et al4 showed that the cpRNFL thickness started to decrease 2 weeks after injury. This was consistent with our study. This discrepancy between scanning laser polarimetry and OCT in the reported onset of reduction in thickness might be related to the technology used.

Macular thickness (as measured with time-domain OCT) also started to significantly decrease at 4 weeks after injury, which was in agreement with previous studies.4,6 In addition, progressive thinning of the GCC was measured using spectral-domain OCT in this study. The GCC loss was greater than macular thickness loss at the final examination (paired t test, P<.001). The GCC reflects structural changes in the inner retina, including the retinal ganglion cells, more accurately than measurements of total macular thickness, which include the outer retinal architecture. The time course of the reduction in GCC thickness was similar to that observed for the cpRNFL. Therefore, the losses of retinal ganglion cells and related axons continue at similar rates as axonal injury. Technological developments that allow for dissociation of the retinal nerve fiber layer from the retinal ganglion cell layer will provide further information.

The current study demonstrated that the reductions in cpRNFL thickness and GCC started to decrease at 2 weeks after trauma and plateaued at 20 weeks in all cases. Certain treatments for TON should be available in the future, but these treatments should be performed by 20 weeks. This study also showed that fragments of the cpRNFL and GCC remained even in eyes that were completely blind. This result might be important during the monitoring of other optic neuropathies such as glaucoma.

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