Optical Coherence Tomography Demonstrates Subretinal Macular Edema From Papilledema

Vincent J. Hoye III, MD; Audina M. Berrocal, MD; Thomas R. Hedges III, MD; Maria Luz Amaro-Quireza, OD

Objective: To evaluate macular changes in eyes with papilledema from increased intracranial pressure using optical coherence tomography (OCT).

Methods: Fifty-five patients with papilledema seen during 1998 and 1999 were studied with OCT of the optic nerve and retinal nerve fiber layer. Nineteen of these also had OCT of the macula during periods of acute, subacute, or recurrent papilledema and were evaluated in detail for this report.

Results: Seven patients had OCT evidence of subretinal fluid involving the macula. All had some reduction in visual acuity. The subretinal fluid appeared to arise from the peripapillary region, and all showed some improvement in central vision as the fluid resolved.

Conclusions: Subretinal fluid accumulations can cause decreased visual acuity in patients with papilledema. Optical coherence tomography can demonstrate subretinal fluid and can be used to follow the course of this important visual complication of papilledema.


Loss of visual acuity from papilledema or optic nerve head swelling due to increased intracranial pressure is uncommon. The visual consequences of papilledema usually are limited to visual field defects and enlargement of the blind spot. Macular abnormalities such as intraretinal macular edema and hemorrhage occasionally cause visual acuity loss in papilledema. Well-documented cases of subretinal macular edema in papilledema are rare in the medical literature. We used optical coherence tomography (OCT), a new technique for imaging the retina, to evaluate the macular region in a group of patients with acute, subacute, and recurrent papilledema.

RESULTS

There was a distinctive space underlying the retina in at least 1 eye of 7 patients who underwent OCT of the macula during an active phase of papilledema. In 2 cases OCT was not evaluated in 1 eye because of a toxoplasmosis scar in one (case 2) and because of recent optic nerve sheath decompression surgery in another (case 3). Five patients were diagnosed with idiopathic intracranial hypertension with normal findings on neuroimaging scans and elevated opening pressures on lumbar puncture. Two patients had brain metastases, one from breast cancer and the other from testicular cancer. Six patients were treated medically with acetazolamide and/or furosemide. One patient received corticosteroids and underwent optic nerve sheath fenestration. The 5 patients with idiopathic intracranial hypertension were followed until there was resolution of the macular subretinal fluid and stabilization of visual acuity (Table).

All 7 patients with OCT evidence of subretinal fluid had some degree of visual acuity loss (<20/25) or visual field loss in one or both eyes. Visual acuities at initial examination ranged from 20/25 to 5/200. Seven of 9 eyes had initial visual acuities of 20/25 to 20/80. The remaining 5 eyes had visual acuities between 20/200 and 5/200 (the eye with toxoplasmosis had a visual acuity of 2/200). Of the 12 patients who had macular OCT findings without evidence of subretinal fluid, visual acuities ranged from 20/15 to 20/20, except for 1 eye in 1 patient in which there was an altitudinal visual field defect.

Final visual acuity, after treatment of the papilledema, ranged from 20/20 to 20/60, with 6 of 11 eyes achieving 20/30
PATIENTS AND METHODS

Optical coherence tomography has been used in our clinic to measure changes in the retinal nerve fiber layer to quantitatively follow the clinical course of papilledema. We have also obtained images of the macular region in many of these patients. In this study, OCT was performed using a prototype instrument using techniques described elsewhere. In addition to circular scans 3.38 mm in diameter around the optic disc, horizontal scans through the macula were obtained in some cases. In some cases the scans were oriented to include the temporal peripapillary space and the macula in the same section. Macular sections were evaluated for the presence of a distinct area of absent reflectivity between the retina and choroid. Macular thickness measurements were made from horizontal OCT sections of the retina.

We reviewed the records of 55 patients with papilledema who underwent OCT of the retina during 1998 and 1999. Most of these patients underwent OCT of the optic nerve and retinal nerve fiber layers. Of these, 19 also underwent OCT of the macular regions when they had acute, subacute, or recurrent optic disc swelling. The rest had chronic atrophic or resolved papilledema or underwent OCT of the optic disc and retinal nerve fiber layer only and were not included in this study. A complete neuro-ophthalmic examination was performed on each patient, including visual acuity, color vision (using AOHRR plates), and automated threshold visual field testing (Humphrey 30-2; Humphrey Inc, Dublin, Calif), except 1 eye that had undergone surgical optic nerve sheath decompression the day previously (case 3). Fluorescein angiography was performed in 2 cases (cases 1 and 2). Lumbar punctures were obtained in all patients except one who had cerebral hemorrhage from brain metastasis, and magnetic resonance imaging or computed tomography was obtained in all cases.

The thickness of the macula and underlying fluid collection in all 12 eyes studied correlated roughly with the degree of visual acuity loss. In the 8 eyes with an initial visual acuity of 20/80 or better, the thickness of the macula and underlying fluid layer measured 370 µm or less (normal macular thickness is 147 ± 17 µm). In the 3 most severely affected eyes, the measurement of macula plus subretinal fluid layer ranged from 510 to 670 µm (case 4, left eye; case 5, both eyes). The eye with the worst vision (left eye, case 5) had the most fluid present. In some cases fluid appeared confined to the macula (cases 1, 4, and 5). However, the others showed apparent communication of subretinal fluid between the macula and the peripapillary subretinal space (cases 2, 3, 6, and 7) (Figure 1D). Visual fields showed enlarged blind spots extending to fixation in 13 eyes tested by perimetry.

In the 12 cases of acute, subacute, or recurrent papilledema in which subretinal fluid was not seen on OCT of the macula, macular thickness measurements ranged from 150 to 210 µm, and visual acuities ranged from 20/20 to 20/50. In those with increased macular thickness greater than 170 µm, there was decreased reflectivity in the papillomacular retina on OCT, but no definite space was seen under the retina in all but 5 eyes. Decreased visual acuity was present in 1 of these cases in which macular thickness was 190 µm. This eye also had significant loss of visual field and a relative afferent pupillary defect consistent with optic neuropathy.

CASE REPORT

A 30-year-old woman with a 4-month history of headache and neck pain developed visual blurring in both eyes. Her visual symptoms consisted of dimming of vision and difficulty with color vision. She also noticed visual phenomena reminiscent of “splinters of glass” in her peripheral vision of both eyes. She weighed 101 kg and was 162.5 cm tall. Corrected Snellen visual acuity was 20/50 OD and 20/60 OS. The pupils were equal and reactive without a relative afferent pupillary defect. Fundus examination was notable for optic nerve edema in both eyes. A peripapillary hemorrhage was present in the right eye (Figure 2A). The macula of each eye showed some irregularity of the light reflex. Automated visual field testing (Humphrey 30-2) showed enlargement of the blind spot in both eyes, an inferior arcuate defect and a paracentral scotoma in the right eye (Figure 2B), and dense superior and inferior arcuate defects in the left eye. Computed tomography of the head showed no abnormality. A lumbar puncture showed an opening pressure of 23 cm H₂O and normal components. In the right eye, OCT demonstrated a space separating the retina from the underlying choroid, suggesting subretinal fluid in the macula of the right eye (Figure 1A). In the left eye, OCT showed thickening of the macula but no definite subretinal space. A fluorescein angiogram revealed no leakage of dye into the macula, only late staining of the optic disc (Figure 2C). The patient was treated with acetazolamide, 500 mg, 3 times daily, and furosemide, 40 mg, twice daily, for 5 weeks. Her visual acuity subsequently improved to 20/25 OU, the papilledema improved, and follow-up OCT dem-
onstrated resolution of the subretinal fluid in the macula of the right eye (Figure 1B).

**COMMENT**

Papilledema causes several different effects on the visual pathways. Chronic papilledema can lead to progressive loss of the optic nerve fibers. This is associated with characteristic arcuate visual field defects, especially in the inferior periphery, along with nerve fiber layer loss more superiorly. Occasionally, loss of central vision may be related to a sudden ischemic episode in an already edematous nerve.

Visual acuity is usually preserved in patients with papilledema. Macular abnormalities that may affect vision include choroidal folds, hemorrhages, or macular retinal nerve fiber layer edema. Retinal pigment epithelial changes consistent with chronic macular edema may also be seen. Additionally, subretinal neovascular membranes may result from papilledema.

Optical coherence tomography demonstrated separation of the retina from the underlying choroid by subretinal fluid in association with decreased visual acuity in 7 cases. In some cases the subretinal fluid appeared to be localized to the subfoveal region (cases 1, 4, and 5) but in others, there appeared be continuity between the subfoveal region and the optic disc, suggesting that the subretinal fluid came from the optic nerve heads (cases 2, 3, 6, and 7). The absence of fluorescein accumulation in the macular region indicates that it did not come from the retinal or choroidal vasculature. In many of the other patients who had OCT of the retina during active papil-

---

**Findings in Patients With Subretinal Fluid From Papilledema**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Sex</th>
<th>Age, y</th>
<th>Affected Eye(s)</th>
<th>Visual Acuity</th>
<th>Test Performed</th>
<th>Diagnosis</th>
<th>Opening Pressure, cm H2O</th>
<th>Thickness of Macula and Fluid Layer, µm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>30</td>
<td>Right (left)</td>
<td>20/50 OD (20/25 OD)</td>
<td>OCT, FA</td>
<td>IIH</td>
<td>23</td>
<td>310 OD (20/60 OD) (20/20 OD)</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>16</td>
<td>Left</td>
<td>20/25 OD (20/20 OD)</td>
<td>OCT, FA</td>
<td>IIH (toxoplasmosis, OD)</td>
<td>38</td>
<td>340 OD (NA, OD) (20/20 OD)</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>24</td>
<td>Right (left)</td>
<td>20/30 OD (Hand motions OS)</td>
<td>OCT</td>
<td>IIH (nerve sheath surgery, OS)</td>
<td>55</td>
<td>310 OD (NA, OS) (20/20 OD)</td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>15</td>
<td>Both</td>
<td>20/50 OD (20/20 OD)</td>
<td>OCT</td>
<td>IIH</td>
<td>50</td>
<td>290 OD (20/60 OD)</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>48</td>
<td>Both</td>
<td>20/40 OD (20/50 OD)</td>
<td>OCT</td>
<td>IIH</td>
<td>28</td>
<td>610 OD (20/60 OD)</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>54</td>
<td>Both</td>
<td>20/30 OD (20/30 OD)</td>
<td>OCT</td>
<td>Metastatic cancer to brain</td>
<td>55</td>
<td>300 OD (20/60 OD)</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>36</td>
<td>Both</td>
<td>20/40 OD (20/40 OD)</td>
<td>OCT</td>
<td>Metastatic cancer to brain</td>
<td>Not obtained owing to hemorrhage</td>
<td>310 OD (20/60 OD)</td>
</tr>
</tbody>
</table>

*OCT indicates optical coherence tomography; FA, fluorescein angiography; IIH, idiopathic intracranial hypertension; and NA (not available), the OCT was not evaluated (owing to toxoplasmosis) or not performed (owing to previous surgery).
illedema in which there was no distinct separation of the retina from the choroid, there was thickening of the maculopapillary retina, suggesting intraretinal edema in these cases. However, this did not seem to affect visual acuity as much as the subretinal fluid appeared to do in the 7 patients in whom OCT showed separation of the retina from underlying choroid.

In 6 cases, we felt that acetazolamide was appropriate initial therapy (cases 1, 2, 4, 5, 6, and 7). All but the 2 patients with metastatic carcinoma responded well, showing resolution of the subretinal fluid and improvement in visual acuity within a few weeks. One patient had more severe and acute visual loss, and we chose optic nerve sheath fenestration in the more severely affected eye as the primary treatment. She received acetazolamide in the postoperative period until resolution of macular edema and papilledema occurred with restoration of visual acuity. Carter and Seiff demonstrated a similar response to optic nerve sheath fenestration in a similar patient.

Subretinal fluid in patients with papilledema was suggested by Reese and, more recently, by Corbett et al as a cause of the enlarged blind spot seen on visual field testing. This idea is supported by histopathologic studies done in 1938 by Samuels, who showed that the intermediary tissue of Kuhnt can become disrupted when there is severe optic disc swelling, allowing for fluid to travel between the retina and the retinal pigment epithelium. Subsequent fluorescein angiography studies suggesting this clinical phenomenon have been reported although in our cases there was no fluorescence of the subretinal space. Horseradish peroxidase studies also indicate that subretinal fluid might arise from cerebrospinal fluid. Although our findings and those of others indicate that subretinal fluid comes from the region of the optic nerve, the exact source of subretinal fluid in papilledema remains uncertain.

We have evidence from OCT to suggest that submacular edema does indeed occur, and, when it does, there may be a direct communication between the subretinal space in the macular region and the swollen optic nerve. Optical coherence tomography shows that this communication may represent an extension of fluid from the optic nerve directly to the macula, and this fluid may cause reduced visual acuity in some patients with papilledema. In our cases the degree of macular thickening was greatest in those patients with the worst visual acuity.

While other macular abnormalities do occur, improvement of visual acuity in our patients after resolution of the subretinal edema further supports a link between visual acuity loss and the presence of subretinal macular edema in some patients with papilledema.

Accepted for publication March 8, 2001.
Corresponding author and reprints: Thomas R. Hedges III, MD, 750 Washington St, Box 381, Boston, MA 02111.

REFERENCES