Subretinal Fluid From Anterior Ischemic Optic Neuropathy Demonstrated by Optical Coherence Tomography

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Objective: To demonstrate the development of subfoveal fluid associated with optic disc swelling from nonarteritic anterior ischemic optic neuropathy.

Methods: Optical coherence tomographic studies obtained during a 3-year period (October 1, 2003, to December 30, 2006) from 76 patients who developed ischemic optic neuropathy from 2 institutions were evaluated. The presence or absence, and the distribution, of subretinal fluid was determined.

Results: Seventy-six patients underwent macular optical coherence tomography within 4 weeks of developing sudden loss of vision in one eye, decreased visual acuity, a visual field defect, a relative afferent pupillary defect, and optic disc swelling with peripapillary hemorrhages. Eight patients had apparent subretinal fluid extending into the subfoveal space. Visual acuity improved in 5 of the 8 patients as the subfoveal fluid resolved.

Conclusions: Subretinal fluid develops in some patients with nonarteritic anterior ischemic optic neuropathy and may contribute to some of the visual loss associated with this condition. Furthermore, resolution of the subretinal fluid could account for some of the visual improvement that can follow anterior ischemic optic neuropathy.


Anterior ischemic optic neuropathy (AION) is associated with optic disc swelling that, in some respects, resembles papilledema from increased intracranial pressure. For example, in addition to obvious distortion of the normal anatomy of the optic disc, in both conditions there is evidence of axoplasmic flow stasis in the optic nerve head.1 Some patients with papilledema develop subretinal fluid that accumulates in the peripapillary region2 and under the macula.3 We have identified evidence of subretinal fluid accumulation in patients who underwent optical coherence tomography (OCT) soon after developing nonarteritic AION (nAION). Most often this occurred in the peripapillary subretinal regions; however, most notably, there was evidence of subfoveal fluid.

METHODS

The records of all patients diagnosed as having nAION from October 1, 2003, to December 30, 2006, in 2 neuro-ophthalmology clinics (86 from the New England Eye Center [NEEC] and 101 from Instituto Nacional de Oftalmologia Ramon Pando Ferrer, Ciudad Habana, Cuba [Drs Gonzalez-Garcia and Mendoza-Santiesteban]) were reviewed. Only patients with typical nAION were selected. All patients were older than 50 years, described sudden onset of unilateral loss of vision, had arcuate or altitudinal visual field defects, had relative afferent pupillary defects, and demonstrated sectoral optic disc swelling. All patients were evaluated within 4 weeks of onset of symptoms. Other causes of optic neuropathy were ruled out by clinical follow-up and additional clinical testing, including erythrocyte sedimentation rates, temporal artery biopsies, or neuroimaging when indicated. During follow-up, those patients who were included in the study had improvement or no change in visual status and developed sector optic nerve atrophy. Patients examined within 4 weeks of the onset of their symptoms included 44 from the NEEC and 59 from the INORPF. Of these patients, all 44 from the NEEC and 32 from the INORPF had undergone OCT. Ten patients from the NEEC and 11 from the INORPF previously had AION in their opposite eyes.

Stratus OCT images (Humphrey; Carl Zeiss Meditec, Dublin, California) were generated using standard scans, 2 mm in axial depth and 6 mm in the transverse direction with resolution of 10 µm axially and 20 µm transversely. The images consisted of 1024 axial pixels and 512 transverse pixels. The pixel spacing was 2 µm per pixel in the axial direction and 12 µm per pixel in the transverse direction. Circumferential (using the fast retinal nerve fiber layer...
Of the 76 patients, 8 had evidence of subfoveal fluid. Of the 44 patients from the NEEC who had OCTs, 9 had peripapillary subretinal fluid only and 19 had peripapillary and subretinal fluid extending toward but not including the fovea.

The clinical findings of the 8 patients with subretinal fluid are summarized in the Table, and the findings from patient 6 are shown in Figures 1, 2, and 3. All patients had acute loss of vision in 1 eye. All had decreased visual acuity and visual field defects suggestive of RNFL damage, including arcuate and/or altitudinal visual field loss. All had relative afferent pupil defects and optic disc swelling with peripapillary hemorrhages. All but 1 (patient 3) had evidence of congenital crowding of the affected optic nerve head and crowding of the contralateral optic nerve head. Other systemic risk factors, such as hypertension, diabetes mellitus, hypercholesterolemia, and sleep apnea, were variably noted; however, none predominated. None had clinical or laboratory evidence of giant cell arteritis; patients 1 and 5 had no histopathological evidence of giant cell arteritis. One patient with similar OCT findings of subfoveal fluid associated with arteritic AION has been observed but is not included in this series.

The OCT findings from the 8 patients with subfoveal fluid included peripapillary subretinal hyporeflectivity next to an elevated optic nerve head (Figure 3). Retinal nerve fiber layer thickening was seen in all 8 patients with average measurements from circular scans and ranged from 268 to 437 µm. Visual acuity reduction roughly correlated with the degree of increased macular thickness (Table). In patients 3, 5, 6, and 8, the subretinal fluid seemed to extend from the optic disc margin toward the fovea, whereas in patients 1, 2, 4, and 7, peripapillary subretinal fluid and an apparently separate area of subfoveal fluid were found, similar to what is seen with central serous maculopathy. In patient 5, who experienced the most severe changes, both peripapillary fluid extending into the macula and a sepa-
rate subfoveal accumulation of fluid were found. A fluorescein angiogram was available for patient 4. It showed staining of the optic disc but no accumulation of dye in or around the macula.

After approximately 1 month, the optic disc swelling clinically resolved and the OCT RNFL measurements showed reduction of RNFL thickness in all patients. The subretinal fluid had resolved and macular thickness measurements returned toward normal in all patients. Visual acuities improved by at least 2 lines in 5 patients.

**COMMENT**

The OCT findings in the 8 patients described herein provide evidence that subretinal fluid that involves the fovea develops in some patients with naION. This finding is similar to the occurrence of subretinal fluid reported in some patients with papilledema. Because we have not
In a previous study of fundus fluorescein angiography did not show subretinal staining or leakage from retinal blood vessels or directly from the choroid. In the macular region, indicating that the fluid did not arise in the retinal space and, in some cases, track into the macular portion of the optic disc. In this situation, subretinal fluid may escape from the peripapillary choroid into the subretinal space in experimental papilledema. Perfusion of vision in nAION might be explained by progressive and reversible effects on the macula by subretinal fluid.

Papilledema and nAION share some pathophysiologic characteristics. For example, in both cases axoplasmic flow stasis and axonal swelling occur. In papilledema, most axonal swelling occurs at the peripapillary region, where the retina is displaced laterally and where serous detachment of the retina may be seen near the optic nerve head. Fluid in the peripapillary subretinal space may accumulate because of disruption of the glial tissues that make up the intermediary tissue of Kuhnt. This is where tracer material has been found to extend from the optic nerve head into the subretinal space in experimental papilledema. Perhaps a similar phenomenon occurs in some patients with nAION, particularly if the infarct involves the temporal portion of the optic disc. In this situation, subretinal fluid may escape from the peripapillary choroid into the subretinal space and, in some cases, track into the macular region. Furthermore, the fluorescein angiogram that was performed in patient 4 did not show accumulation of dye in the macular region, indicating that the fluid did not arise from retinal blood vessels or directly from the choroid. In patients with subretinal fluid from papilledema, fluorescein angiography did not show subretinal staining or leakage. In a previous study of fundus fluorescein angiography did not show subretinal staining or leakage. In 5 of our patients with nAION, visual acuity improved substantially as the subretinal fluid resolved. In 2 patients, there was only slight improvement, and in 1 patient with severe nAION, no improvement was found. In some patients with nAION the visual loss may be progressive, and in many individuals visual acuity may improve. If subretinal fluid is present in such patients, the pattern of progression and improvement of vision in nAION might be explained by progressive and reversible effects on the macula by subretinal fluid.

Submitted for Publication: April 15, 2007; final revision received June 13, 2007; accepted June 14, 2007.

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Financial Disclosure: None reported.

Funding/Support: This study was supported in part by an RPB Challenge grant (New England Eye Center/Department of Ophthalmology, Tufts University School of Medicine).

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